DISSERTATION ON

A STUDY OF PAROTID SWELLINGS

Submitted to

THE TAMILNADU

DR.M.G.R.MEDICAL UNIVERSITY

in partial fulfillment of the requirement

for the award of degree of

M.S.DEGREE EXAMINATION

BRANCH – I

GENERAL SURGERY

KILPAUK MEDICAL COLLEGE AND HOSPITAL

THE TAMILNADU

DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI

APRIL 2013

CERTIFICATE

This is to certify that **"A STUDY ON PAROTID SWELLINGS"** is bonafide of record done by **Dr.ANU RAMESH,** in the Department of General Surgery, Kilpauk Medical College and Hospital, Chennai-10 during the post graduate course from 2010-2013 under the guidance and supervision of **Prof.P.N.SHANMUGASUNDARAM, M.S.,** in partial fulfillment for the award of M.S. DEGREE EXAMINATION, BRANCH-I (GENERAL SURGERY) to be held in April 2013 under Tamilnadu Dr M.G.R. Medical University, Chennai.

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DECLARATION

I declare that this dissertation entitled **"A STUDY ON PAROTID SWELLINGS"** is a record of work done by me in Department of General Surgery, Kilpauk Medical College, Chennai-10 during my Post Graduate course from 2010-2013 under the guidance and able supervision of my unit Chief and Head of Department, Department of General Surgery **Prof.Dr.P.N.SHANMUGASUNDARAM M.S.,** it is submitted in partial fulfillment for the award of M.S. DEGREE EXAMINATION-BRANCH I (GENERAL SURGERY) to be held in April 2010 under the Tamilnadu Dr.M.G.R. Medical University, Chennai. This record of work has not been submitted previously by me for the award of any degree or diploma from any other university.

Dr. Anu Ramesh

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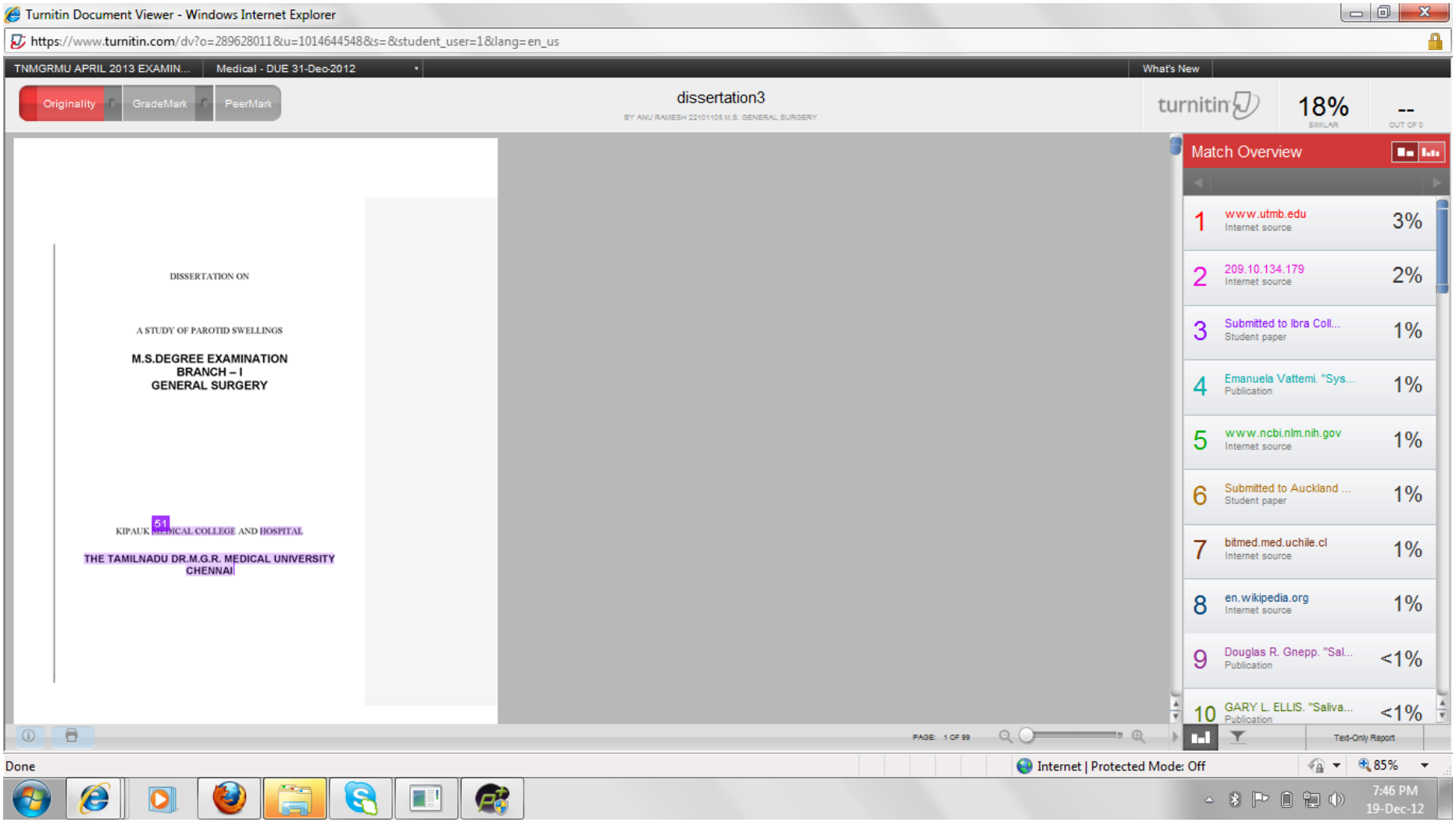
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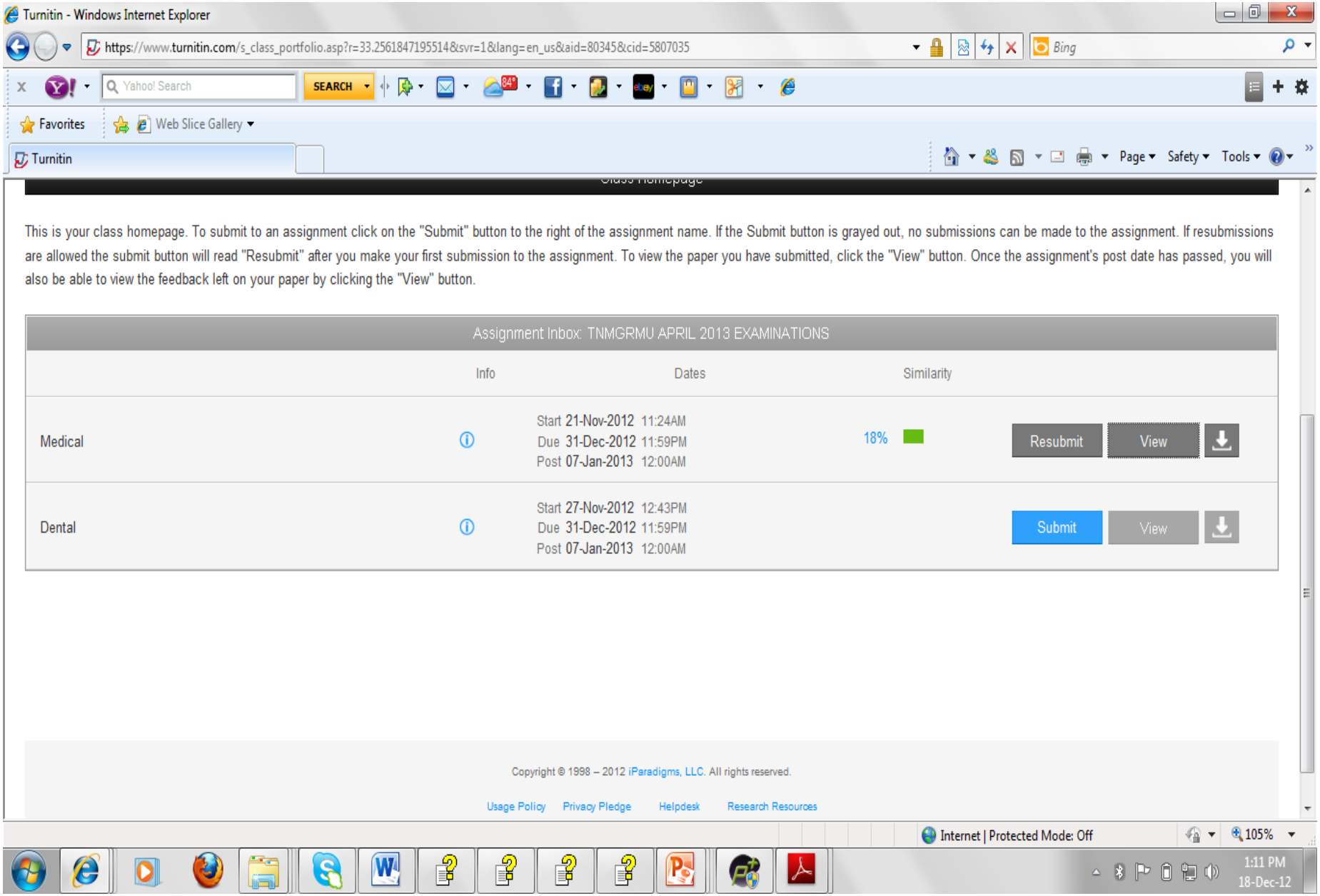
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ABSTRACT

INTRODUCTION:

Swellings of the parotid gland are of special interest to a surgeon's keen eye. These lesions are not only involved in diseases isolated to the parotid but can also present as a part of a generalized systemic disorder, medical or surgical. For a surgeon the interests lie in the probable origin of the swelling, its involvement of the facial nerve, the variability in behavior, regarding the operability criteria and its post-operative complications. A comprehensive knowledge of the anatomy of the parotid and the prediction of the swelling behaviour can help not only in the diagnosis but also in ensuring an apt management of the lesion and the patient.

AIMS:

This cohort study was conducted to analyse the following in our institution

1. The incidence of various of parotid swellings.
2. To discuss accuracy of FNAC in comparison to the histo-pathological reports.
3. The various surgical modalities of treatment of parotid swellings applied .
4. To discuss the post-operative complications.
5. To compare findings of the above study with world statistics.

MATERIALS AND METHODS:

The cohort study which included 45 patients was conducted at Kilpauk medical college hospital and Government Royapettah Hospital from September 2010 to October 2012. Data was collected from the patients after obtaining an informed consent. The demographic details of the patients and history of their

swelling was taken. The patients were examined and basic investigations performed. Details regarding the FNAC report, surgical and non-surgical management were noted. Post-operative complications were documented. The final histopathological report was analysed and compared with the FNAC report. Inclusion criteria were patients with parotid swellings neoplastic and non-neoplastic and those above 12years of age. Exclusion criteria were patients with parotid lesions due to systemic or metabolic illness and those with age less than 12 years.

CONCLUSION:

The analysis of the data of the study conducted at our institution provided us with the following results:

1. Parotid lesions comprised of the most common salivary gland lesions in our hospital.
2. Amongst the various lesions it was noted that benign tumours were the most common and the least common were non-neoplastic disorders.
3. The sex incidence showed a similar distribution among both males and females with the ratio being 1:1.25.
4. The mean age of presentation was 49 yearsand it was seen that the 4th and 7^{th} decades where the predominant age group for occurrence in case of benign and malignant tumours respectively.
5. The lesions which were predominant in the non-neoplastic, benign and malignant tumours groups where abscess, pleomorphic adenoma and mucoepidermoid carcinomas respectively. These were found to be consistent with the comparison made with world statistics.
6. FNAC correlated in a total of 39 out of 45 cases, i.e.86.67% of the cases.

The sensitivity and specificity for detection of benign tumors was found to be 93.75% and 100% respectively. In the case of malignant tumours the sensitivity and specificity was found to be 87.5% and 100% respectively.

7. Patients presenting with facial nerve palsy was seen more amid the malignant tumors.
8. Most commonly performed surgery was superficial parotidectomy. Completion parotidectomy was performed in 2 cases and both were malignant tumors with recurrence.
9. Facial nerve palsy and seroma formation were the commonest complication noted post-operatively.
10. Radiotherapy was the most common non-surgical modality used and administered more commonly post-operatively.

CONTENTS

INTRODUCTION

Swellings of the parotid gland are of special interest to a surgeon's keen eye. These lesions are not only involved in diseases isolated to the parotid but can also present as a part of a generalized systemic disorder, medical or surgical. The patient would present invariably due to the cosmetic problem. For a surgeon the interests lie in the probable origin of the swelling, its involvement of the facial nerve, the variability in behavior, regarding the operability criteria and its post-operative complications. Patients in these cases present themselves to oncologists and general surgeons alike for the same. A comprehensive knowledge of the anatomy of the parotid and the prediction of the swelling behaviour can help not only in the diagnosis but also in ensuring an apt management of the lesion and the patient.

REVIEW OF LITERATURE

"It follows from the complex relations of the parotid that its entire removal as a surgical procedure is an anatomical impossibility."

Sir Frederick Treves, Surgical anatomist

The parotid gland derives its name from the Greek word meaning "para auricular" swelling. Surgical treatment of parotid tumors has been greatly influenced by the intimate relationship of the facial nerve to the gland. The intricacy of its anatomy to the neuro-vascular structures has plagued anatomists and surgeons alike for years. McWhorter[13] considered that the gland was divided into two lobes, with an interconnecting isthmus and the facial nerve passing around the isthmus and between the two lobes. The consensus has changed over the years to believe that the gland actually is divided into deep and superficial portions due to the emergence of the facial nerve. The parotid duct was discovered in 1660 by Niels Stensen and hence named after him[1]. The first clinical description of a parotid tumour was given in 1752 by Kaltshmeid, whereas a classification of tumors was given by Berard in 1841. For several years the parotid gland surgeries were performed with dismal results with respect to the facial nerve preservation. In 1892, the first attempt at total parotidectomy with facial nerve preservation was done by Codreanu [1]. It was Blair in 1912 with careful dissection observing keenly for facial twitching while stimulating the nerve fibres devised the

technique for tumor removal with nerve preservation. But it was Sistrunk in 1921 who stressed the importance of exposure of the facial nerve while dissection of the gland[1]. It has been seen with several studies the commonest lesions affecting the parotid are the group of benign tumours. Incidence of non-neoplastic lesions has been found to be rarer in comparison, varying with studies and inflammatory disorders being the most common. A R Arshad, et al [16] have seen that the incidence of non-neoplastic lesions was found to be around 11.8% where as Zbaren et al[15] have seen an incidence of 5.7%. Pleomorphic adenoma has been seen to be the dominant lesion affecting the parotid. The treatment of pleomorphic adenoma has undergone a vast change from the initially performed enucleation of the tumour to the present concept of parotidectomy, superficial or total based on the depth of the tumors. Malignant parotid tumours are treated based on the lesion, its extent of involvement and attempts at preserving the facial nerve, unless it is involved, is the key concept at present. The concept of change to nerve preservation has brought down morbidity due to the nerve paralysis significantly improving on the cosmesis and quality of life of patients. An understanding of the cause-effect relationship of parotid tumours is still under research though many theories have been proposed regarding them.

EMBRYOLOGY:

The development of the parotid gland is mostly seen around the sixth week of development, as an ectodermal derivative, when the formation of the duct occurs. It appears as an outgrowth from the oral epithelium and covers the first branchial arch's maxillary process. It then grows posteriorly towards the ear and hence embedding the facial nerve along with its branches within the substance of the gland. There is then formation of canals from the solid cords and the differentiation of cells from the tips leads to formation of acini which are secretory in nature.

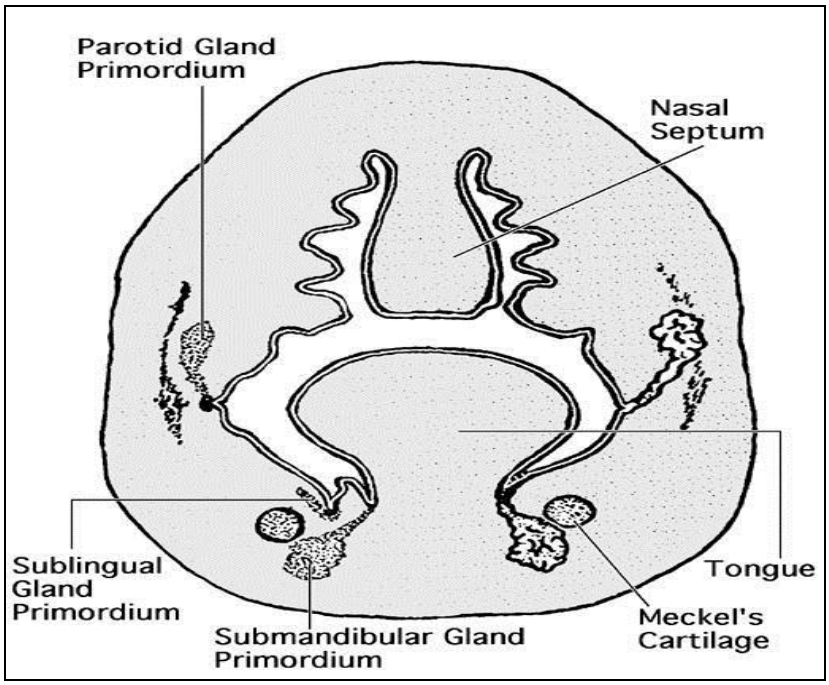


Fig. a) DEVELOPMENT OF PAROTID GLAND

ANATOMY:

The parotid gland is the largest of the three salivary glands and is paired. It occupies the parotid region, named so due to the presence of the gland, which is present from below and front of the ear to below the zygomatic arch. It has the advantage of an irregular shape hence fitting snugly in the space provided for it. The gland is divided due to the presence of the facial nerve in to superficial (endo facial) and deep (exo facial) parts, around 80% of the gland being superficial. It shares its space in the compartment with the facial nerve and its branches, external carotid artery and its terminal branches, retro mandibular (posterior facial) vein and its divisions, lymph nodes and sensory and autonomic nerves. The compartment boundaries comprise of the following:

Anterior Border	Diagonal line drawn from zygomatic root to the External auditory canal
Posterior Border	External auditory canal
Superior border	Zygoma.
Inferior Border	Styloid process, styloid process musculature, internal carotid artery, jugular veins

The superficial part lies over the masseter and mandible, while the deep part or retro-mandibular portion extends through the

stylomandibular tunnel medially, which is in turn formed by posterior edge of the ramus(ventrally), anterior border of sternocleidomastoid and posterior belly of digastric (dorsally) and also the stylomandibular ligament deep dorsally. The stylomandibular ligament in fact separates the parotid and submandibular glands. The deep part of the gland is seen in the prestyloid compartment of the Parapharyngeal space and hence tumors in this portion of the gland can push the tonsillar fossa and soft palate medially in the intra-oral part. The gland is enclosed within a capsule continuous with the deep cervical fascia; the layer covering the superficial surface is dense and closely adherent to the gland forming the false capsule. A portion of the fascia, attached to the styloid process and the angle of the mandible, is thickened to form the stylomandibular ligament. True capsule is formed from condensation of the fibrous stroma of the gland. The superficial part is somewhat quadrilateral in shape, being broad above and tapering somewhat below

Five processes have been described in the gland of which three are said to be superficial while two are deep. The superficial processes are:

1. Condylar 2.Meatal 3. Posterior

The deep processes are

1. Glenoid 2. Stylomandibular

Due to the presence of multiple processes it is extremely difficult to ensure complete clearance of the gland during surgeries and this difficulty is increased due to the presence of the facial nerve.

The gland has five surfaces, the apex, base or superior surface, superficial surface, anteromedial and posteromedial surfaces.

The three borders of the gland are anterior, posterior and medial.

Relations of the parotid:

The antero-medial surface is molded on the posterior border of the ramus of the mandible and clothed by the medial pterygoid and masseter. The other anterior relations are the lateral surface of the temporo-mandibiular joint and the emerging branches of the facial nerve.

The postero-medial surface is grooved longitudinally and is seen to hitch against the external auditory meatus, the mastoid process and anterior border of sternomastoid. Thus its relations comprise of the mastoid process with the sternomastoid, posterior belly of digastric, styloid process and its attachments, external carotid artery which enters the gland through this surface. The internal carotid artery is deep to the styloid process in this part.

The superficial surface slightly lobulated is covered by the integument, the superficial fascia containing the facial branches of the

great auricular nerve, platysma muscle and some small lymph glands, and the parotid fascia which forms the capsule of the gland. Parotid lymph nodes are embedded within the gland substance.

The superior surface or the base is related to the cartilaginous portion of the external auditory meatus, temporo-mandibular joint, superficial temporal vessels and auriculo-temporal nerve. The auriculo-temporal nerve winds around the neck of the mandible.

The apex of the gland is seen to overlap the posterior belly of digastric and carotid triangle. Emerging from the apex are the two divisions of the retro-mandibular veins and the cervical branch of facial nerve.

The borders of the gland are anterior, posterior and medial. The anterior border has the following structures emerging from it: the parotid duct, terminal branches of the facial nerve and transverse facial vessels. The medial border is present such that it separates the antero-medial and postero-medial surfaces. The posteromedial and superficial surfaces are separated by the posterior border.

The process of the gland:

Glenoid process is in the superior part of the gland extends upwards into the posterior mandibular fossa. The facial process is in the anterior margin of the gland extending forwards into the masseter.

The pterygoid process is seen between the medial pterygoid muscle and the mandibular ramus.

Intra-glandular structures are(from medial to lateral):

1. External carotid artery and its terminal branches

2. Retromandibular vein

3. Facial nerve and its terminal branches

The external carotid artery lies at first deep to the gland and then within the gland. In its substance it gives its two terminal branches, the internal maxillary and the superficial temporal arteries. The former runs forward deep to the neck of the mandible; the latter runs upward across the zygomatic arch and gives off its transverse facial branch which emerges from the front of the gland.

Lateral to the artery the corresponding venous branches internal maxillary and superficial temporal unite to form the retro-mandibular vein (posterior facial vein). In the lower part of the gland this vein splits into anterior and posterior divisions. The anterior division emerges from the gland and unites with the anterior facial to form the common facial vein; the posterior unites in the gland with the posterior auricular to form the external jugular vein.

The most superficial of the structures is the facial nerve. The branches of which emerge from the borders of the gland. The course of the facial nerve after its exit from the stylomastoid foramen begins with branches being given off to the stylomastoid, posterior belly of digastric and posterior auricular muscles before its transition to formation of pes anserinus which occurs about 1cm anterior and 2 cm below the tragus. The facial nerve enters the gland from its postero-medial surface. It gives of its terminal branches from divisions:

1. Temporo-facial – temporal, zygomatic and buccal branches

2. Cervico-facial–marginal mandibular and cervical branches.

The facial nerve and the retro-mandibular vein divide the gland into superficial and deep parts by the Facio-venous plane of Patey. Several studies regarding the facial nerve branching pattern have been done and the following conclusions were made[2, 3, 4].

Type I- No anastomosis occurred between branches of the facial nerve.

Type II- Presence of an anastomotic connection between branches of temporo-facial division.

Type III-A single anastomosis between the temporo-facial and cervico-facial divisions

Type IV- Combination of Type II and Type III.

Type V- Two anastomotic rami passed from the cervico-facial division to intertwine with the branches of temporo-facial division.

Type VI- Plexiform arrangement, the mandibular branch sends a twig to join any members of the temporo-facial division.

Studies have shown variation in frequency of the type of facial nerve branching pattern. Weerapant et al[2] compared their results with other groups and found that Type V was the most predominant while Type I was the least common. Others such as Davis et al[3] showed that Type III was the most common type.

Among the other nerves encountered in relation to the parotid the greater auricular nerve is the first to be encountered during raising of the skin flaps and is commonly sacrificed in surgery leading to anaesthesia of the region. The auriculo-temporal nerve is a branch or the trigeminal nerve and it relays post ganglionic parasympathetic secreto-motor fibres to the parotid to from the otic ganglion.

The identification of facial nerve is of critical importance.

The following are the important surgical landmarks for the same[5]:

1. Tragal pointer- points to the main trunk of VII nerve proximal to the Pes and is 1–1.5 cm deep and below the pointer.

2. Tympano-mastoid suture – when traced medially the main trunk can be encountered 6–8 mm deep to the suture line.

3. Posterior belly of digastric muscle – It is a guide to the stylo-mastoid foramen; the trunk of the VII nerve is just superior and posterior to cephalic margin of the muscle.

4. Styloid process- 5–8 mm deep to the Tympano-mastoid suture; trunk lies on the postero-lateral aspect of the Styloid near its base

5. By retrograde dissection one of the branches can be traced proximally

    a. Buccal branch- it runs with the parotid duct either superiorly or inferiorly.

    b. Temporal branch- crosses the zygomatic arch parallel with the superficial temporal artery and vein

    c. Marginal Mandibular branch - runs along the inferior border of the parotid superficial to the retro-mandibular vein.

The Stylomastoid foramen is the single most constant landmark.

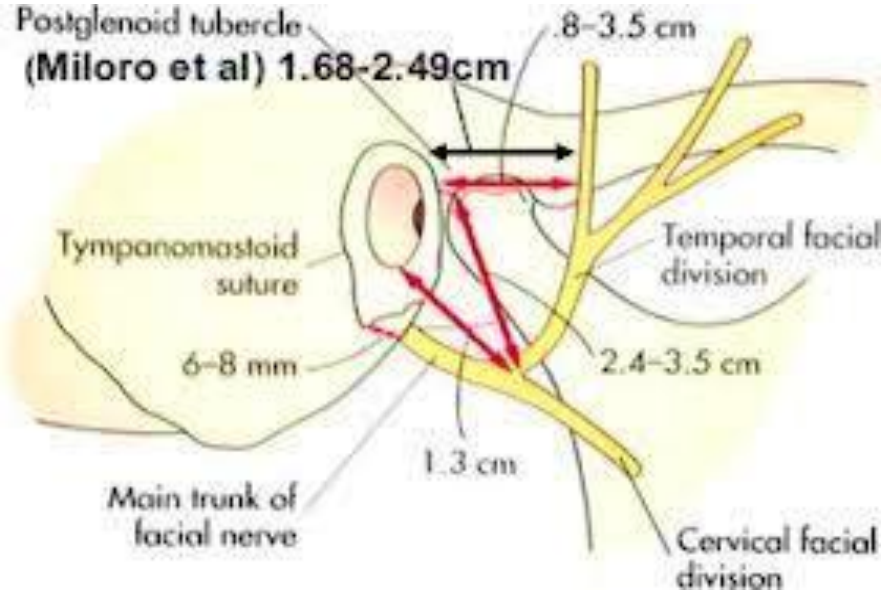


Fig.b) IDENTIFICATION OF FACIAL NERVE INTRA-OPERATIVELY WITH LANDMARKS

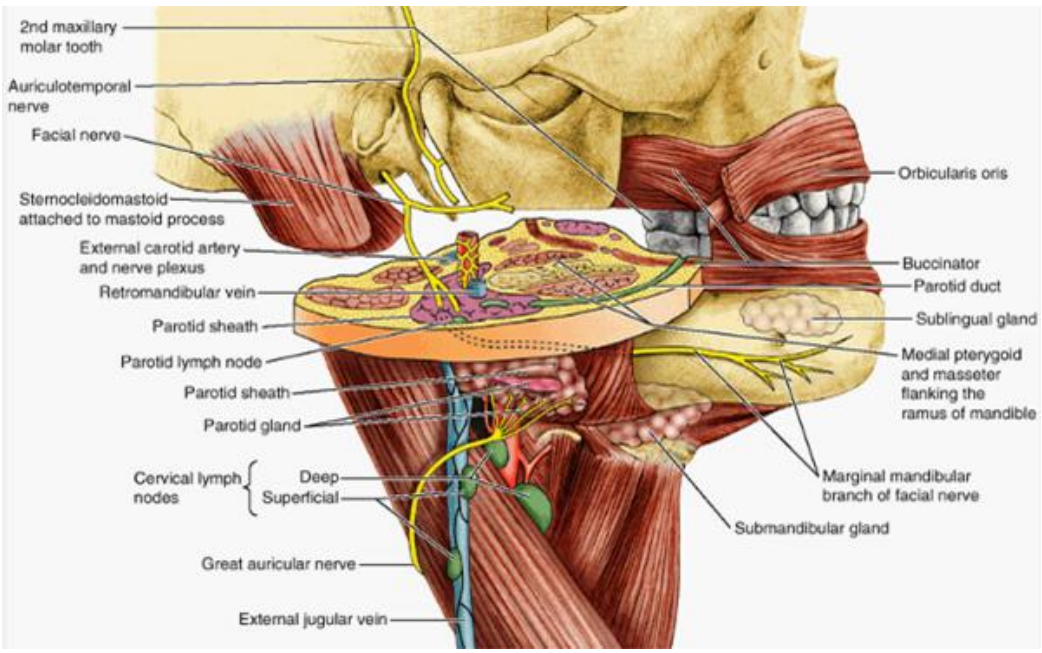
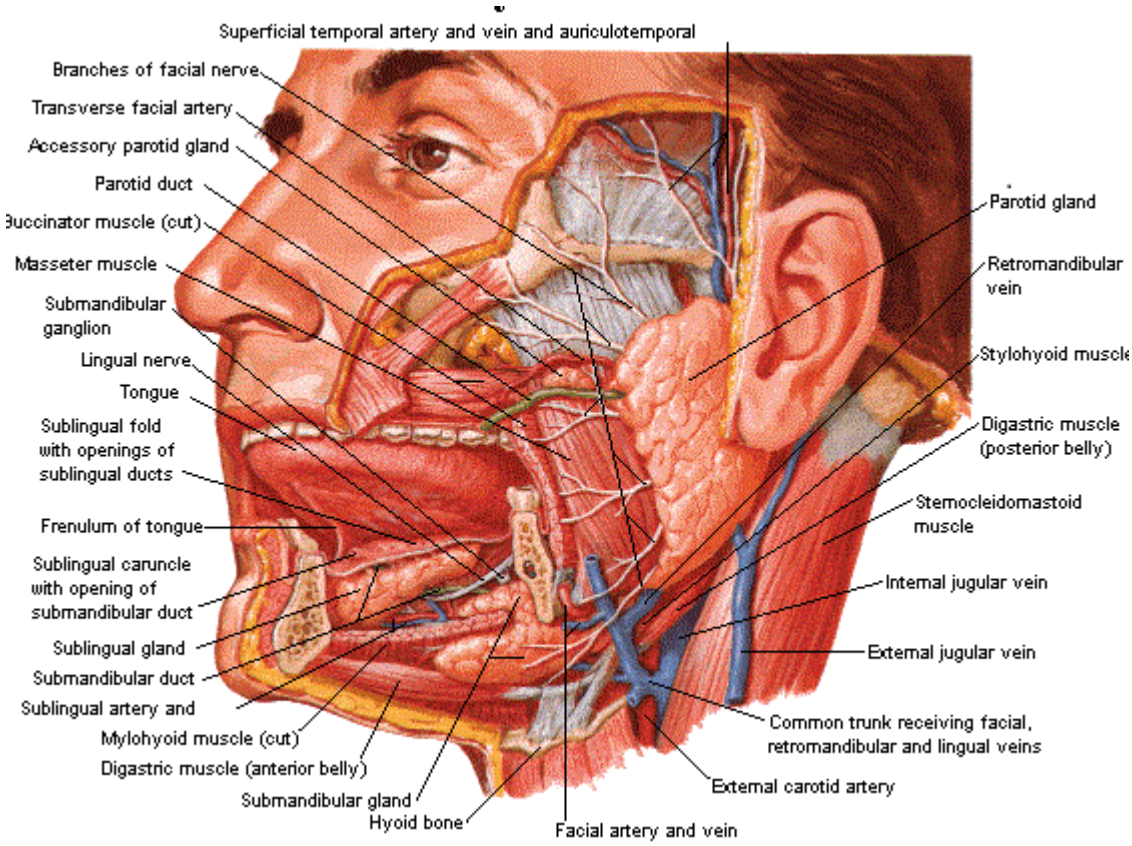


Fig.c) STRUCTURES RELATED TO PAROTID GLAND (SECTION TAKEN THROUGH THE GLAND)



Fig,d) ANATOMY OF PAROTID GLAND WITH FACIAL NERVE BRANCHES

Stenson’s duct:

It arises from the anterior border of the parotid and parallels the zygomatic arch, around 1.5cm below the inferior margin. It runs superficial to the masseter muscle and turns medially 90 degrees to pierce the buccinator muscle at the level of the second maxillary molar where it opens onto the oral cavity. It measures approximately 4–6 cm in length and 5mm in diameter. The buccal branch of the VII nerve runs along with it.

The Parotid is invested in its own fascia (capsule), which is continuous with the superficial layer of deep cervical fascia. The Parotid fascia consists of

1) Superficial layer – extends from the masseter and Sternomastoid to the Zygoma, and

2) Deep layer – extends from the fascia of the posterior belly of the Digastric muscle, and forms the Stylomandibular membrane separating the Parotid and Submandibular glands.

The Parotid fascia sends septa into the glandular tissue, which prevents the possibility of separating the glandular tissue from its investing fascia. The attachments of the Parotid fascia include

Anteriorly	Mandible
Inferiorly	Stylomandibular ligament
Posteriorly	Styloid process

Vascular supply:

It is supplied by the branches of the external carotid artery and drained by external jugular vein.

Nerve supply:

It is via the Auriculo-temporal nerve which is a branch of the mandibular branch of trigeminal nerve.

The parasympathetic nerves are secreto-motor. The preganglionic fibres begin in the inferior salivary nucleus; pass through the IX nerve, its tympanic branch, the tympanic plexus and the lesser petrosal nerve and relay in the otic ganglion. The post ganglionic fibres pass through the auriculo-temporal nerve and the gland.

Sympathetic nerves are vasomotor and are derived from the plexus around the external carotid artery.

Sensory nerves are from the auriculo-temporal nerve to the gland but the parotid fascia is innervated by the sensory fibres of the greater auricular nerve.

Lymph nodes: The parotid nodes lie partly in the superficial fascia and partly deep to deep fascia over the parotid. This occurs as during development the parotid is the last to get encapsulated hence results in encapsulation of the nodes. Also during this process salivary epithelial cells can be included within the nodes and believed to play a role in development of Warthin's tumor[5].

HISTOLOGY:

The parotid gland is a predominantly serous salivary gland contains numerous serous acini. Also there are zymogen granules, intercalated and striated ducts. Small lymph nodes within the gland give rise to interstitial lymphocytes. Varying degrees of adipocytes are also seen depending upon the patient's age.

The parotid gland secretion is watery and is about 20% of the total secretion from salivary glands.

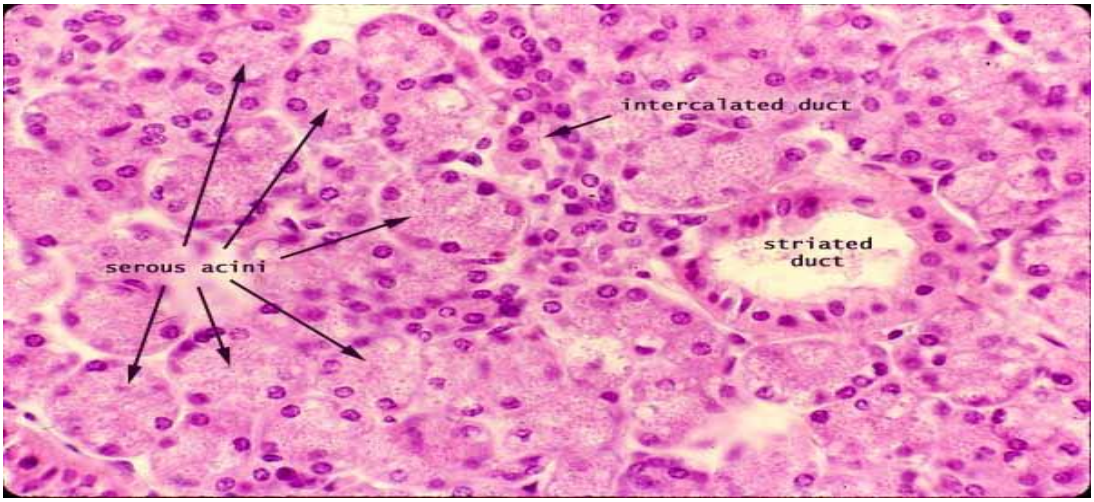


Fig.e)HISTOLOGY OF PAROTID GLAND

PAROTID LESIONS:

Non-neoplastic disorders of the parotid can be classified as follows[7, 17]:

1. Inflammatory
    * Acute (specific)
        a. Viral (mumps, Coxsackie virus A, echovirus, and lymphocytic choriomeningitis)

        b. Bacterial (staphylococcal, streptococcal, pneumococcal, Gram-negative)

Acute suppurative of infancy

Postsurgical

Terminal debilitation

* Chronic (specific)

  Tuberculosis

  Actinomycosis

  Sarcoidosis

* 'Recurrent subacute' and chronic recurrent

  Self-limited

  Progressive

  Lymphoepithelial lesion and Sjogren's syndrome

2. Systemic and Secondary Metabolic Disorders

3. Obesity, hypertension, diabetes mellitus, malnutrition and associated deficiencies (proteins, vitamins), alcoholic liver disease

   Hypersensitivity and Drug idiosyncrasy
   Local (Salivary Gland) Disturbances

   Sialolithiasis

   Sialoangiectasis

   Trauma, foreign body, fistula

   Parotid lymphadenopathy

Cysts, mucocele and ranula

Local duct obstructions (mucous plugs, congenital)

4. Miscellaneous

Pneumoparotitis

Psychogenic

Functional over activity

Idiopathic

Irradiation sialadenitis

NON-NEOPLASTIC PAROTID LESIONS :

1. Parotitis :

These are seen to occur due to bacterial, viral causes or as a result of auto-immune disorders. Viral parotitis is the most common and caused by paramyxovirus. Other viral infections associated are Epstein-Barr virus, Coxsackie and parainfluenza virus. Bacterial infections associated are Staphylococcus aureus and Streptococcus viridans. Bacterial infections lead to abscess formation. Antibiotics and if necessary incision and drainage are the mainstay of treatment in the above cases.

Autoimmune disorders such as Sjögren's syndrome, rheumatoid arthritis and hypergammaglobulinemia. In early part of autoimmune

disorders lymphoplasmocytic infiltrate is seen with almost no parenchymal distortion. If a nodular collection > 50 lymphocytes (“ focus score”) is seen, the condition is said to be chronic sialadenitis[20]. Late in the disease, near complete absence of acini is noted along with minimal ducts and dense intra-epithelial lymphocytosis. These conditions can later on lead to lymphomas.

2. Sialolithiasis

This is seen less in the parotid gland in comparison to the sub-mandibular gland. They result from the concretions that coalese within the duct system. It can secondarily lead to chronic sialadenitis. It is a painful condition associated with increased pain and swelling during meal times. The stone if radio-opaque can be visualized in radiographs. Treatment involves removal of the stone and gland portion which is affected.

3. Chronic Sialadenitis (Chronic Sclerosing Sialadenitis):

It is usually a unilateral condition which occurs most commonly due to an obstructive sialolithiasis; other causes are radiotherapy or duct strictures. It is also called Kuttner’s Tumour and can clinically mimic a neoplasm. Histology will show dilated, secretion filled ducts with lymphoplasmocytic infiltrate with occasional germinal centre early in the disease. In late stages, fibrotic ducts surround the gland with acinar atrophy. Treatment is by surgical excision.

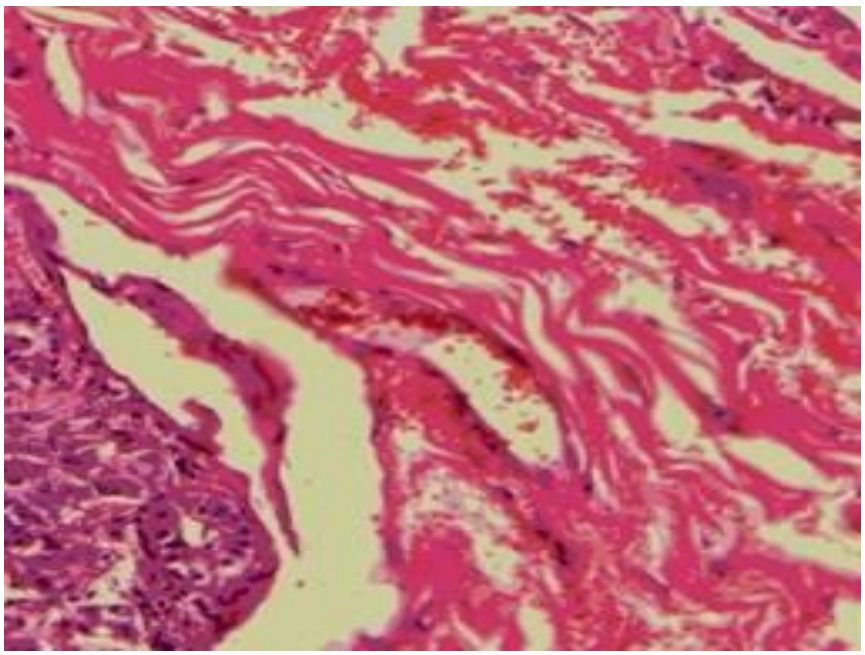


Fig.f) HISTOPATHOLOGY OF CHRONIC SIALADENITIS

NEOPLASTIC DISORDERS:

The parotid gland is known for the high preponderance of benign tumors such as pleomorphic adenoma which is the commonest lesion. WHO classification of parotid neoplasms.

TABLE - 1
WHO CLASSIFICATION OF PAROTID NEOPLASMS[9]

MALIGNANT TUMOURS	BENIGN TUMORS
Acinic cell carcinoma	Pleomorphic adenoma
Mucoepidermoid carcinoma	Myoepithelioma
Adenoid cystic carcinoma	Basal cell adenoma
Polymorphous low-grade adenocarcinoma	Warthin tumor
Epithelial-myoepithelial carcinoma	Oncocytoma
Clear cell carcinoma, not otherwise specified	Canalicular adenoma
Basal cell adenocarcinoma	Sebaceous adenoma
Sebaceous carcinoma	Lymphadenoma- sebaceous and non-sebaceous
Sebaceous lymphadenocarcinoma	Ductal papillomas - Inverted ductal papilloma -Intra ductal papilloma Sialadenoma pappiliferum
Cystadenocarcinoma	Cystadenoma
Low-grade cribriform cystadenocarcinoma	
Mucinous adenocarcinoma	**SOFT TISSUE TUMOURS**
Oncocytic carcinoma	Haemangioma
Salivary duct carcinoma	
Adenocarcinoma, not otherwise specified	**HAEMATOLYMPHOID TUMOURS**
Myoepithelial carcinoma	Hodgkin lymphoma
Carcinoma ex pleomorphic adenoma	Diffuse large B-cell lymphoma Extranodal marginal zone B-cell lymphoma
Carcinosarcoma	
Metastasizing pleomorphic adenoma	**SECONDARY TUMOURS**
Squamous cell carcinoma	
Small cell carcinoma	
Large cell carcinoma	
Lymphoepithelial carcinoma	
Sialoblastoma	

The exact etiology of parotid lesions is unknown but several factors have been implicated including environmental and genetic. Over time, certain risk factors and clarification of causes have been done

Proof of cause and effect does not exist in any of these postulated associations, and the etiology of most salivary gland cancers cannot be determined

ETIOLOGY OF NEOPLASTIC LESIONS:

J.W. Eveson et al[9] study on neoplastic lesions proposed the following causes.

1. VIRUSES:

Strong associations between lympho-epithelial carcinomas and Epstein - Barr virus (EBV) have been made[9].

2. RADIATION:

Evidence to understand compelling links between ionizing radiation and parotid tumors have been studied. Follow-up on a long term basis of atomic bomb victims showed contributory evidence towards an increase in these tumors. Those undergoing therapeutic radiation for the head and neck tumors also have an increased risk.

3. OCCUPATION:

Industrial workers such as those in rubber manufacturing and plumbing industry due to exposure to metal and nickel compounds are prone for parotid tumors. Also those individuals in the woodworking, automobile industries and employed in asbestos mining are prone for increased risk of parotid tumors.

4. LIFE STYLE :

Though no association has been found between alcohol consumption, a definite association is found between Warthin's tumor and smoking. An increased level of risk has also been postulated in those with high cholesterol intake.

5. HORMONES:

Conflicting reports regarding associations of endogenous hormones in parotid tumors have been reported. Estrogen receptors were found in nearly 80% of normal glands in males and females. Estrogen receptors have been reported in a minority of cases of acinic cell carcinoma, mucoepidermoid carcinoma and salivary duct carcinoma, but not detected in adenoid cystic carcinoma[9]. Certain studies have also reported estrogen receptors in pleomorphic adenoma.

Progesterone receptors are noted with high levels of expression in recurrent pleomorphic adenoma. Among malignant lesion, they were seen acinic cell carcinoma, adenoid cystic carcinomas and mucoepidermoid carcinomas.

Androgen receptors are present in 90% of salivary duct carcinomas. A recent study has also shown this immune-reactivity in carcinoma ex pleomorphic adenoma and basal cell adenocarcinoma and a fifth of cases in the study showed positivity in acinic cell carcinoma, mucoepidermoid carcinoma and adenoid cystic carcinoma.

PAROTID TUMORS:

BENIGN TUMORS:

1. Pleomorphic Adenoma-

It is also known as benign mixed tumor as originally said by Minssen in 1874 and comprises of multiple histologic components including myxoid, mucoid, chondroid and other elements, hence known for its heterogeneity. These comprise almost 80% of parotid neoplasms[9]. To distinguish from a malignant transformation, features such as cellular atypia, mitosis, perivascular and perineural invasion are relied upon. They are slow growing, painless tumors, arising in 90% of cases from the superficial lobe. These tumors are noted to have pseudopods and hence have a tendency to recur if only enucleated.

The epithelial component consists of ductal structures with an associated myoepithelial layer, but also may contain collections of myoepithelial cells that may be spindled, clear, plasmacytoid, or basaloid. The mesenchymal, or stromal, component is typically myxoid, hyaline, or chondroid

Pleomorphic adenomas have been divided into a myxoid type (>80% mesenchymal-type tissue), cellular type (>80% epithelial-type tissue), and mixed or classic type (generally an equal mix of components)[20]. Treatment is surgical and superficial parotidectomy is done in most cases. Total parotidectomy is done for deep lobe involvement.

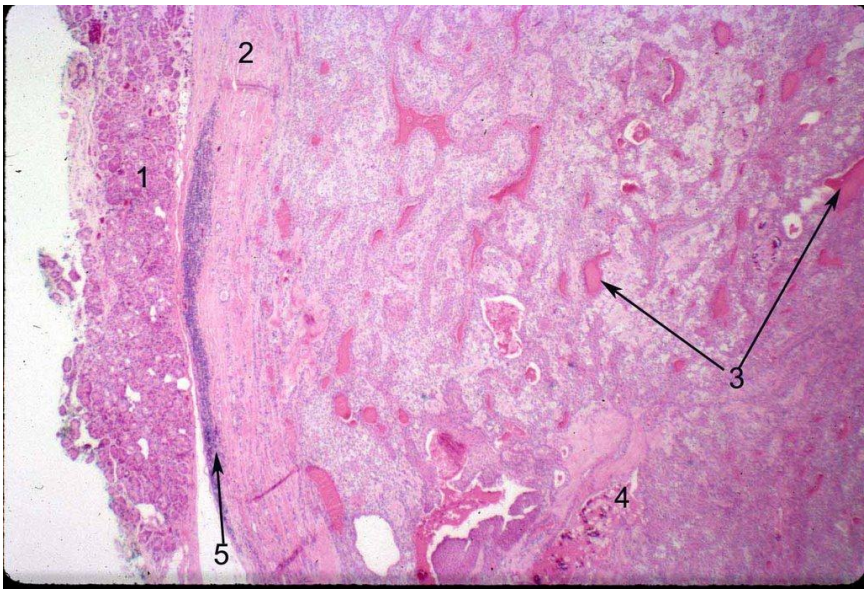


Fig.g) MICROSCOPIC PICTURE OF PLEOMORPHIC ADENOMA

2. Warthin’s tumor:

This is also known as papillary cystadenoma lymphomatosum. It is the second most common benign parotid neoplasm associated with a10% bilateral incidence, male predominance and multi-centricity. Also it is almost exclusively found in the parotid gland. As mentioned earlier the late encapsulation of the gland and lymphatic tissue trapping during development favours the formation this tumor. Another salient feature of this tumor is that it contains plenty of mitochondrial rich oncocytes and hence presents as hot spots on radio nucleotide with Technetium 99M[21]. Microscopically what we see characteristically are two-tiered epithelial layer, lining the branching, cystic or cleft-like spaces and immediately subjacent, well-developed lymphoid tissue sometimes forming germinal centers[20].

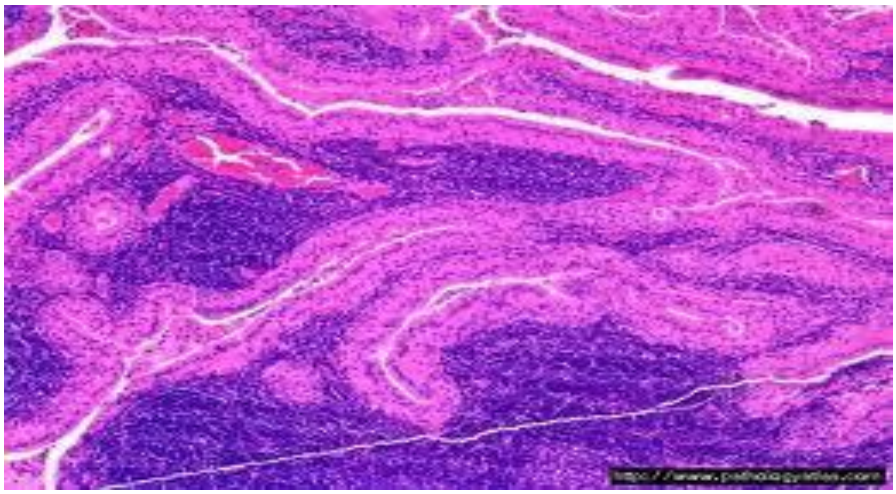


Fig. h)MICROSCOPIC PICTURE OF WARTHIN'S TUMOUR

3. Basal cell adenoma:

They are benign tumors basaloid cells. They tend to occur generally in adults with 75% occurring in the parotid gland. They are usually asymptomatic, slow growing lesions. A subtype called the dermal anlage tumor may be multicentric and may be associated with various adenexal skin tumors. Though four patterns are seen microscopically, the tumor is composed of 2 cell types. Small cells with little cytoplasm typically lie at the neoplasm's edge, frequently show peripheral palisading, and give the tumor its basaloid appearance. The other type being more polygonal basaloid cells with slightly more cytoplasm and round to oval nuclei containing more open chromatin usually lay in the tumor's center. They are immunopositive for pan-cytokeratin S-100, smooth muscle actin, and muscle-specific actin, all evidencing myoepithelial differentiation.

4. Myoepithelioma :

These are benign tumors which are almost exclusively composed of myoepithelial cells although a small percentage can be made up of ductal cells. Hence they are considered to lie at one of the spectrum with basal cell adenoma at the opposite end and pleomorphic adenoma more in the center[20]. It is seen to occur in almost equal frequency in the parotid gland and minor salivary glands. Histologically the lesion comprises of

sheets and cords of tumor cells. They are classified into four sub-types all of which have collagenous or myxoid stroma:

I) Spindle-cell II) Hyaline III) Plasmatoid

IV) Clear cell

Cells stain strongly positive for S-100 and cytokeratin and are variably reactive to smooth muscle actin and glial fibrillary acid protein (GFAP)[20].

MALIGNANT TUMORS:

1. Mucoepidermoid carcinoma:

It is the most common parotid malignancy, composed of mucous, intermediate, and epidermoid (or squamoid) cells. They are noted to be slightly more common in women with a mean incidence around the 5th decade of life[20]. Patients present with a slow growing, painless mass. Hallmark of these tumors is the presence of three different cell types which can occur in sheets, nests, duct like structures or cysts. Frequently intermediate cells predominate, ranging from small basal cells with minimal basophilic cytoplasm to larger oval cells with pale eosinophilic cytoplasm. The mucin-producing cells are organized singly or in clusters , with pale, foamy cytoplasm, distinct cell membranes, and eccentric small nuclei. They line cystic spaces and are positive with mucicarmine or PAS stains[20]. Abundant eosinophilic cytoplasm and vesicular nuclei are seen in epidermoid or squamoid cells. Immunohistochemistry is of little utility in

the diagnosis of mucoepidermoid carcinoma. Many mucoepidermoid carcinomas possess a t (11;19)(q21;p13) translocation[20]. Tumors that carry the rearrangement are associated with a better clinical outcome. Prognosis is highly dependent on the grade of the tumor. Low-grade lesions are markedly cystic, and have abundant well-differentiated mucous cells. High-grade lesions are more solid with squamous and intermediate cells predominating.

Table - 2

BRANDWEIN GRADING OF MUCOEPIDERMOID CARCINOMA[20]:

PARAMETER	POINTS
Cystic component<25%	2
Tumor front invades in small Nests and islands	2
Pronounced nuclear atypia	2
Lymphatic and or vascular invasion	3
Neural invasion	3
Necrosis	3
4+ mitosis/ 10 HPF	3
Bony invasion	3

Table - 3

GRADING OF MUCOEPIDERMOID CARCINOMA

Grade	Point score/mortality (%)
Low (I)	0
Intermediate (II)	2–3
High (III)	4 or more

Wide local resection has to be done for these tumors. Radiation can be used in cases of recurrence or for palliation in case of unresectable lesions. Prognosis depends upon the grade of the tumor, excellent for low grade (>90%) and poorer for high grade tumor (about 50%)[20].

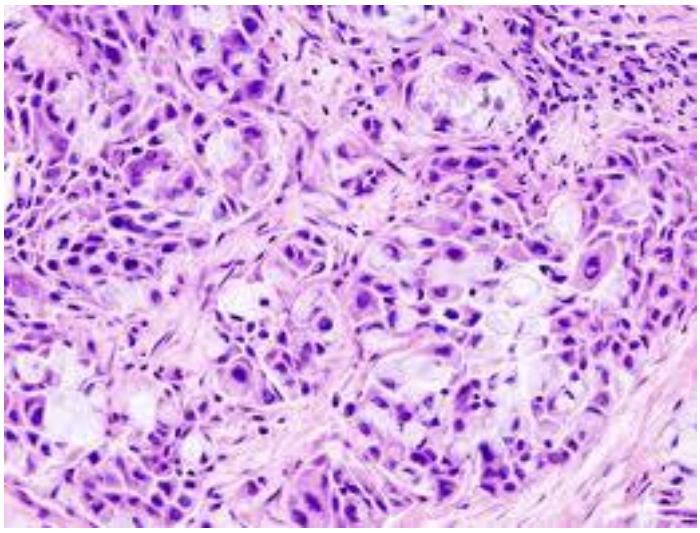


Fig.i) MICROSCOPY OF MUCOEPIDERMOID TUMORS

2. ACINIC CELL CARCINOMA:

It accounts for a total of 1%–3% of salivary gland tumors of which it's most common location is the parotid. It is the second most common malignancy[20]. It presents as a slowly growing mass, can be occasionally painful. It is seen usually as a single, circumscribed, solid mass and can undergo cystic degeneration. Histological variability is seen. It can be solid or lobular, microcystic, papillary-cystic or follicular. Small tumors due to their well-differentiated state can be missed easily. The characteristic cell seen is the acinic cell, which has the appearance of a salivary acinar cell with abundant granular, basophilic cytoplasm and a small, round, eccentrically placed nucleus. With PAS staining cytoplasmic zymogen granules are seen. A mixture of architectural patterns is seen and characteristic dense lymphoid infiltrate is also seen. Due to lack of tumor infiltration at the tumor periphery it can be confused as benign. Differential diagnosis includes a normal parotid gland, oncocytoma, clear cell carcinoma, cystadenocarcinoma. Adequate resection is necessary as recurrence is seen in about one- third of cases. It is considered as a low-grade malignancy, but around 10%–15% metastasizes regionally to lymph nodes or in a distance to lungs and bones. Survival rate is around 80% in 5 years[20].

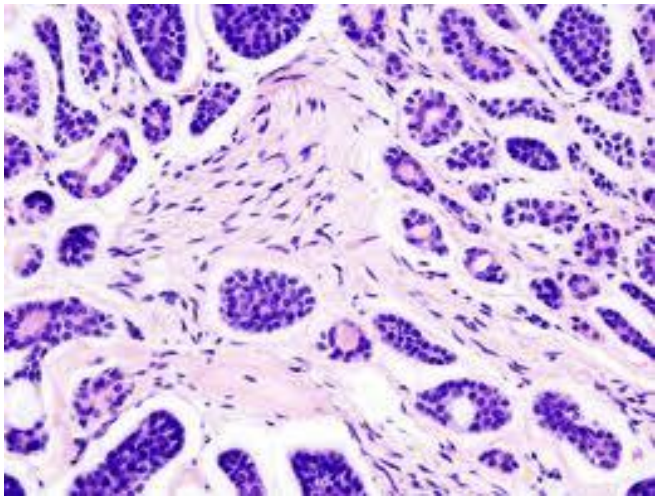


Fig.j) MICROSCOPIY OF ACINIC CELL CARCINOMA

3. ADENOID CYSTIC CARCINOMA:

Peak incidence of this tumour is seen in patients between 40 and 60 years of age. It is slow growing and progressive in nature. Perineural invasion is characteristic to this disease and at times it can be the presenting symptom of the disease such as facial nerve palsy. Varying architectural patterns can be seen, cribriform, tubular, solid and mixed. Grading is based on the dominant pattern; most commonly seen is cribriform, consisting of cell nests arranged around gland-like spaces filled with PAS positive granular basophilic material. The spaces are actually extra-cellular cavities containing reduplicated basal lamina and myxoid material. The tumor cells are basaloid with round to oval, hyperchromatic nuclei without nucleoli and very little cytoplasm.

Grading of Adenoid Cystic Carcinoma

Predominant pattern	Grade
Tubular	I
Cribriform	II
Solid	III

Perineural invasion is common in the tumor periphery. Immunohistochemistry is of little use. Positive reaction for cytokeratins, collagen type IV and laminin and partial reactivity towards myoepithelial markers is seen. This tumor is highly malignant and progressive. Compared to other cancers this has a lower survival rate of around 62% at five years[20]. Involvement of bone, perineural invasion and solid type of tumors show poorer prognosis.

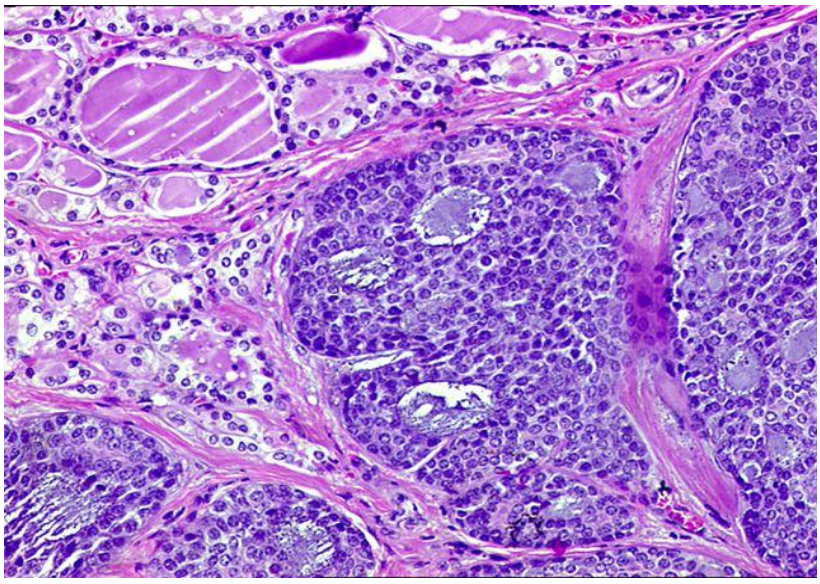


Fig.k) MICROSCOPIC PICTURE OF ADENOID CYSTIC CARCINOMA

4. MALIGNANT MIXED TUMORS:

This term is broadly used to true malignant mixed tumors, carcinoma ex pleomorphic adenoma and metastatic mixed tumor[20].

I) True salivary gland mixed tumor(carcinosarcoma)

This is composed of both carcinomatous and sarcomatous components and is extremely rare, About one third of patients have pre-existing pleomorphic adenoma. It presents around the sixth decade and microscopically it is seen to have an intimate admixture of both components. High grade duct carcinoma or undifferentiated carcinoma mixed with fibrosarcoma, leiomyosarcoma or liposarcoma is seen[20]. These are extremely aggressive tumors treated with wide local excision n and radiotherapy.

II) Carcinoma ex pleomorphic adenoma:

These account for >95% of mixed malignant tumors. The classical history is a long standing parotid mass that has undergone rapid growth over few months. The risk of malignancy increases with the number of years the tumour is left untreated, 1.5% in 5 years and 9.5% in 10 years[21]. The proportions of carcinoma and pleomorphic adenoma can vary; the malignant component can be poorly differentiated adenocarcinoma, salivary ductal carcinoma or undifferentiated carcinoma. Prognosis is dependent upon the carcinoma type and extent of invasion. Invasion is

classified as intracapsular (noninvasive), minimally invasive (≤ 1.5mm in the greatest extent) or invasive (≥1.5mm in the greatest extent) [20]. Wide resection with lymph node dissection in cases of nodal metastasis is done and radiotherapy provided postoperatively or for invasive and inoperable tumours.

III) Metastasizing mixed tumor:

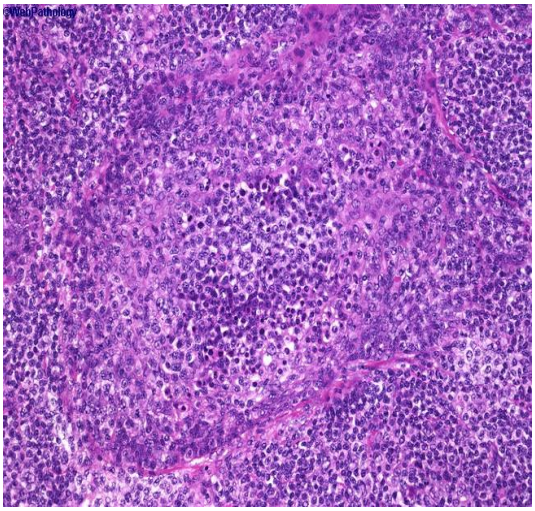
This is the least common. Histologically they resemble pleomorphic adenoma but are associated with metastasis to local lymph nodes or distant organ metastasis.

5. SALIVARY DUCT CARCINOMA:

This is one of the most aggressive primary salivary gland tumors. This resembles high-grade ductal carcinoma of breast. Male are more commonly affected and clinically it seen as a rapidly growing parotid mass with skin and facial nerve involvement. These are well demarcated and partly encapsulated with invasion into adjacent parenchyma. Microscopy shows ductal structures lined by eosinophilic cells with supporting layer of clear myoepithelial cells with hyalinized, eosinophilic stroma between cells. Wide local excision and radiotherapy are the treatment modalities.

6. LYMPHOMA:

It is extremely rare and can occur in < 5% of patients with parotid lesions. Suggestive clinical features are development of a parotid mass in a known patient of malignant lymphoma, or suffering from an immune disorder (Sjogren's syndrome, rheumatoid arthritis or AIDS) or in an individual with a previous benign lymphoepithelial lesion. The prognosis is usually better than nodal lymphoma of same histology.



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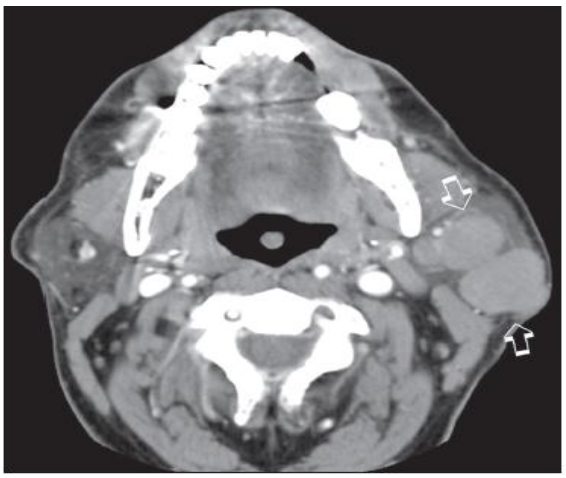


B

Fig. l) LYMPHOMA OF PAROTID GLAND -A. MICROSCOPIC PICTURE B. CT PICTURE

7. METASTASIS TO PAROTID:

As such the metastasis is more to the intra or periglandular lymph nodes and they are most commonly from the primary tumors of the head and neck. The parotid nodes provide the drainage basin from the scalp, face, ear skin, external auditory canal and tympanic membrane. Hence squamous cell carcinomas and melanomas account for around 80%. The remainder is most commonly from the lung, kidney and breast carcinomas.

PHYSICAL EXAMINATION:

The diagnosis depends upon essential findings in the history and physical examination. In the examination one should focus on the extent of disease in the parotid, neck, local effects of the lesion and nerve involvement. The mass is palpated to determine presence of pain, its consistency, mobility and fixity to the adjacent tissue. The skin of scalp, face and ear is examined for lesions. Any evidence of neck node involvement is palpated for. Oral cavity is examined for the duct opening and deep lobe assessment. The pharyngeal wall is examined for deviation and jaw is examined for trismus. Findings suggestive of malignancy include a large, fixed mass, facial nerve weakness, nodal metastasis, skin involvement and at times trismus.

Fig.m) PAROTID GLAND TUMOUR

Fig.n) PAROTID LESION SEEN IN THE TAIL REGION

STAGING OF PAROTID TUMORS

TNM staging

PRIMARY TUMOUR:

TX	Primary tumor cannot be assessed
T0	No evidence of primary tumor
T1	Tumor ≤2 cm in greatest dimension without extra parenchymal extension
T2	Tumor >2 cm but not >4 cm in greatest dimension without extra parenchymal extension
T3	Tumor >4 cm and/or tumor having extra parenchymal extension
T4a	Tumor invades skin, mandible, ear canal, and/or facial nerve
T4b	Tumor invades skull base and/or pterygoid plates and/or encases carotid artery

REGIONAL LYMPH NODES (N)

NX	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastasis
N1	Metastasis in a single ipsilateral lymph node, ≤3 cm in greatest dimension

N2a Metastasis in a single ipsilateral lymph node, >3 cm but not >6 cm in greatest dimension

N2b Metastasis in multiple ipsilateral lymph nodes, none more than 6 cm in greatest dimension

N2c Metastasis in bilateral or contralateral lymph nodes, none >6 cm in greatest dimension

N3 Metastasis in a lymph node, >6 cm in greatest dimension

DISTANT METASTASIS (M)

MX Distant metastasis cannot be assessed

M0 No distant metastasis

M1 Distant metastasis

Table 4: **STAGE GROUPING- AJCC**

STAGE GROUPING			
Stage I	T1	N0	M0
Stage II	T2	N0	M0
Stage III	T3	N0	M0
Stage III	T1	N1	M0
Stage III	T2	N1	M0

Stage III	T3	N1	M0
Stage IVA	T4a	N0	M0
Stage IVA	T4a	N1	M0
Stage IVA	T1	N2	M0
Stage IVA	T2	N2	M0
Stage IVA	T3	N2	M0
Stage IVA	T4a	N2	M0
Stage IVB	T4b	Any N	M0
Stage IVB	Any T	N3	M0
Stage IVC	Any T	Any N	M1

PROGNOSTIC FACTORS:

1. Age at diagnosis 2. Pain at presentation 3. T stage
4. N stage 5. Skin invasion 6. Facial nerve dysfunction
7. Perineural growth 8. Positive surgical margins
9. Soft tissue invasion 10. Treatment type

MANAGEMENT

INVESTIGATIONS:

FINE NEEDLE ASPIRATION:

Cytological analysis which can be achieved through FNA is helpful in a pre-operative evaluation to distinguish between malignant and benign lesions. It is considered safe when done with 23 gauge needle. It can also be done with a 25 gauge needle[11.]

Most commonly performed blindly in the outpatient clinic and has several advantages – it is quick, safe and accurate in the hands of a skilled practitioner and high levels of diagnostic accuracy. Accuracy increases when used under ultrasound guidance[12]. An average accuracy is estimated around 54% to98%. The diagnostic accuracy is less in cases of malignant lesions compared to benign ones. Sensitivity up to 70% and specificity around 94% can be obtained. It can be useful to distinguish not only between benign and malignant tumor, but also salivary and non-salivary processes. It can be indicated to identify suspected malignancies, diagnose metastatic carcinomas, suspected lymphomas and evaluate bilateral tumors. Also it helps enable conservative management in Warthin's tumour or pleomorphic adenomas in poor risk patients. A study by A.M. Contucci et al[26] where the FNA and final histopathology were compared showed sensitivity and specificity of 57.3% and 100% respectively with and overall diagnostic accuracy of 94%.

RADIOLOGICAL INVESTIGATIONS:

1. SIALOGRAPHY:

This helps in diagnosing lesions of the Stenson's duct such as strictures, calculi. Also in the presence of any parotid enlargement the displacement of the duct can be seen. It is rarely used, both because there is a significant possibility of an infectious flare-up and because the study actually yields only minimal information.

2. ULTRASOUND;

It helps to distinguish between solid and cystic lesions and also to identify lymph nodes. An important application is when it is used in guided FNAC increases the accuracy of FNAC.

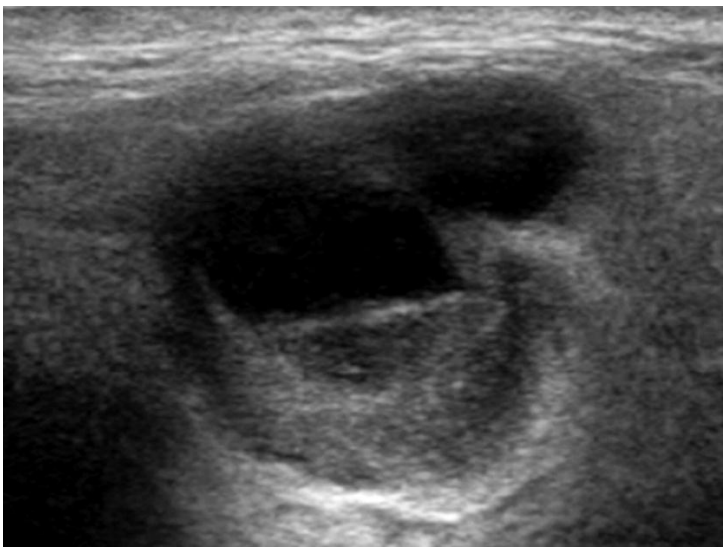


Fig.o) ULTRASOUND OF PAROTID LESION- CYSTIC APPEARANCE

A study published regarding ultrasound guided FNA showed that in 74 out of 76 cases a cytological diagnosis was achieved[22].

3. CT

CT is superior to MRI for evaluation of the bony structures. It is indicated in patients with diffuse enlargement of the parotid gland, tumor extension beyond the superficial lobe, facial nerve weakness, trismus, or deep-lobe parotid tumors that are difficult to evaluate clinically[10]. If the parotid mass appears to be fixed to the deeper structures, it is appropriate to proceed with CT to evaluate the extent and parapharyngeal extension of the disease.

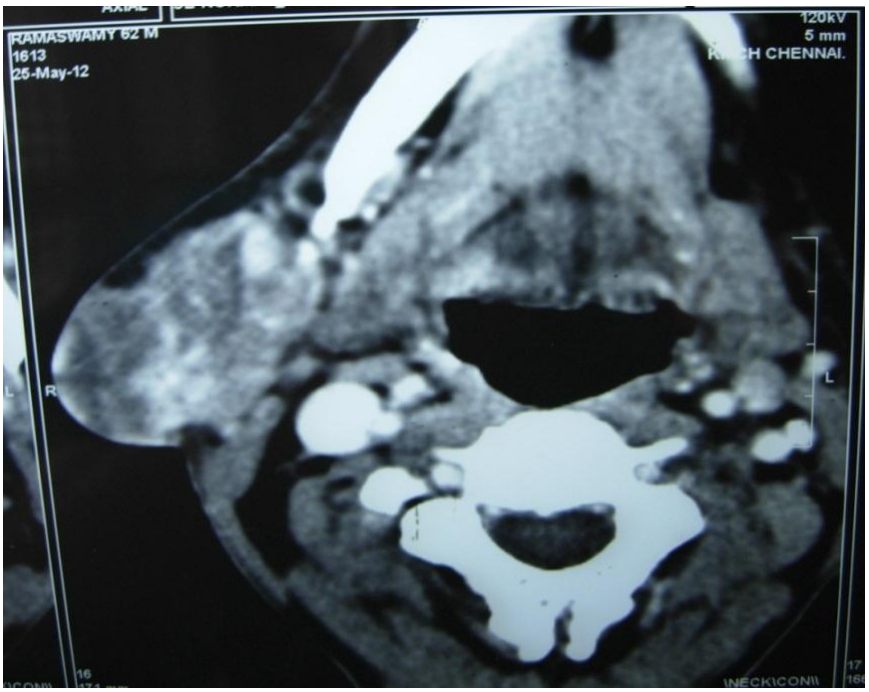


Fig.p)CT PICTURE OF PAROTID NEOPLASM

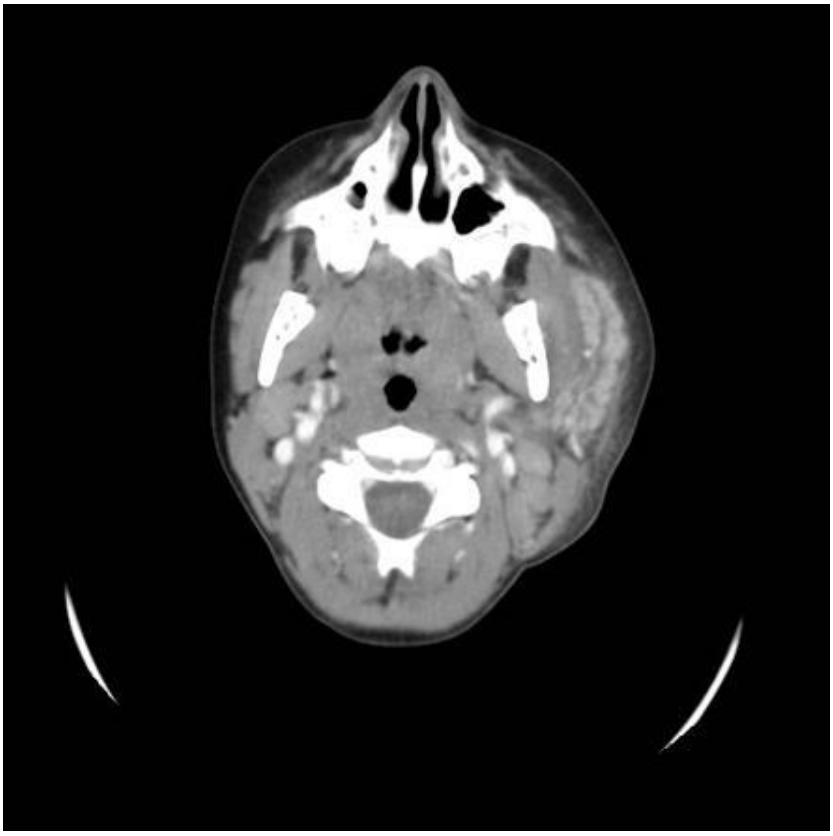


Fig.q)CT PICTURE OF ACUTE PAROTITIS

MRI:

MRI helps to distinguish inflammatory lesions from neoplasms of the parotid. It is indicated in cases of facial nerve palsy. It also is helpful in distinguishing deep lobe tumors from other parapharyngeal masses, evaluating suspicious lymph nodes and the periphery of the mass

RADIONUCLEOTIDE TECHNITIUM 99M SCAN:

This is seen to be helpful in identifying Warthin's tumors and oncocytomas as hot spots that they present with due to the presence of oncocytes. They are not used presently as before.

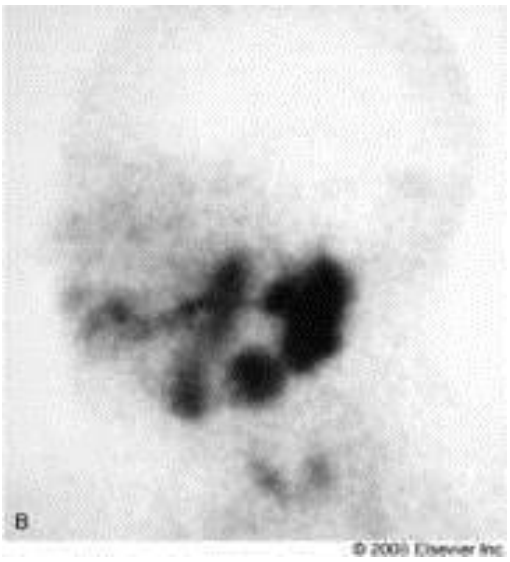


Fig.r) RADIONUCLEOTIDE SCAN IN ONCOCYTOMA

TREATMENT:

Surgery is the primary treatment done for most of the parotid swelling presenting in a Surgical department. Other modalities which may be used are radiotherapy as an adjuvant or neo-adjuvant form. Chemotherapy is administered in cases of palliation or lymphomas.

SURGICAL:

Principles of treatment of parotid lesion[10]:

1. Adequate local excision of tumor, based on extent of primary lesion and the primary lesion itself.

2. Preservation of facial nerve if possible

3. Elective neck dissection reserved for selected patients

4. 4.Postoperative radiotherapy when indicated (in appropriate fields)

5. Prognosis determined primarily by stage and grade of tumor.

The basic types of surgeries done for removal of the parotid gland are as follows:

1. Superficial parotidectomy
2. Total conservative parotidectomy
3. Radical parotidectomy

1. SUPERFICIAL PAROTIDECTOMY:

It is the minimal surgical procedure for a parotid mass requiring removal involving the superficial lobe. Identification and dissection of the facial nerve is of extreme importance as otherwise inadvertent injury can occur. General anaesthesia without muscle relaxation should be advocated for this. Incision for the procedure should be planned keeping the adequate surgical exposure and cosmesis in mind. It begins anterior to the ear, just above the tragus, continuing downward past the tragus, curving behind the ear and turning downward to descend along the sternocleidomastoid. The incision is deepened upto the subcutaneous level and a plane is developed between the external ear canal and the posterior aspect of the parotid. Anterior flap is created in a plane superficial to the parotid fascia. The sternomastoid muscle after

identifying its anterior border is retracted after dissecting the gland from it. The Greater auricular nerve may be sacrificed if required. Dissection should be continued along the plane and attachments to the mastoid be cut and the posterior belly of digastric muscle is identified. Dissection should be done in a vertical plane to minimize risk of injury to the distal branches of facial nerve[10]. At this point the identification of the facial nerve should be made as it emerges from the stylomastoid foramen. The nerve can be identifies by an antegrade or a retrograde approach. In the antegrade approach the main trunk of the nerve is identified, usually located at a point which underlies the halfway point between mastoid process tip and ear canal. Other landmarks include tragal pointer, the posterior digastric belly and tympanomastoid suture. To ensure the safety of the nerve, several centimeters of the parotid need to be mobilized. At a proximity to the nerve electrocautery is best avoided. Retrograde approach is used when the main trunk cannot be exposed and hence dissection is proceeded from a peripheral branch to the main trunk. Alternatively nerve stimulator may be used to identify the branches. Once the nerve trunk has been identified and dissected all around carefully, sharp dissection is used to divide the gland substance while protecting the nerve. It is important that each division of the gland reveals more of the nerve. Once the dissection is done, all the branches of the nerve have been defined and the superficial part of the gland removed, after ensuring hemostasis the wound can be closed with a suction drain in situ. The patient is closely monitored for any facial nerve palsy.

2. TOTAL PAROTIDECTOMY

This is employed in case of malignant lesions and lesions with deep lobe involvement. After proceeding as superficial parotidectomy the nerve trunks are gently retracted and excision of the deep lobe is done. In case there is retromandibular extension of the tumor, the incision can be extended anteriorly over the mentum and paramedian mandibulotomy can be performed.

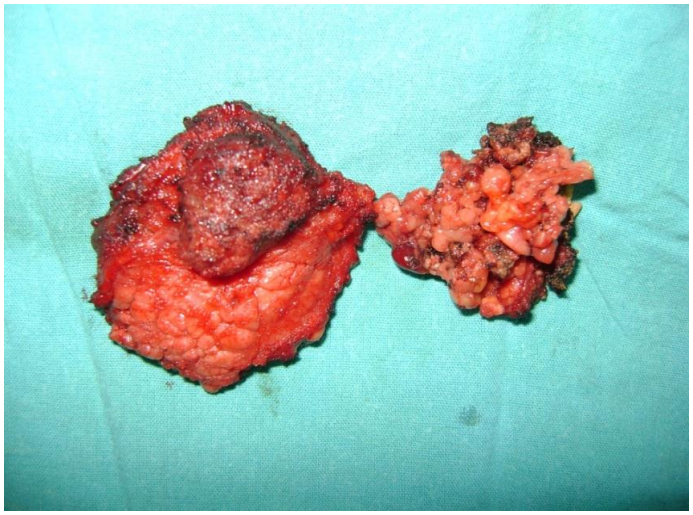


Fig. s) SPECIMEN OF TOTAL PAROTIDECTOMY

3. RADICAL PAROTIDECTOMY:

It is employed for patients with advanced parotid carcinomas. Facial nerve is sacrificed. Extended radical surgery involves resection of overlying skin, adjacent mandible and soft tissue, temporal bone and portion of the adjacent external ear. Free tissue transfer used for repair of facial nerve.

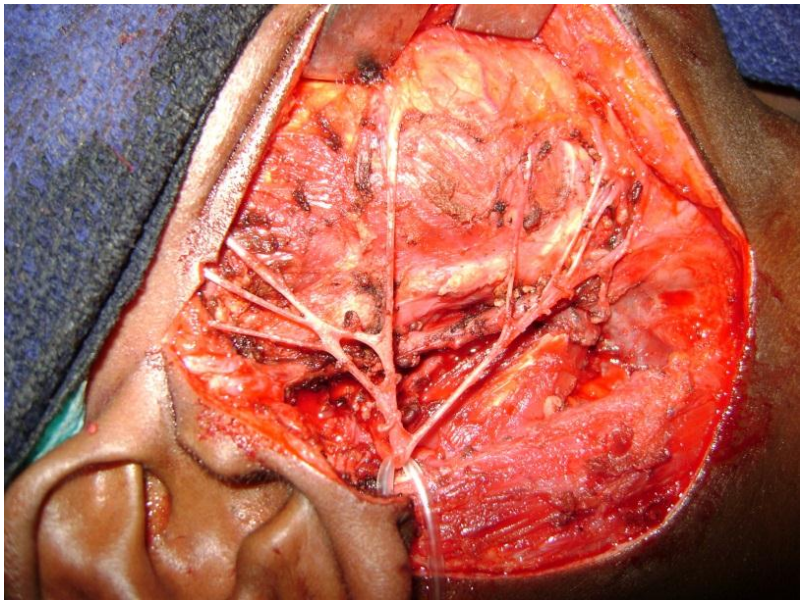


Fig.t) INTRA-OPERATIVE FACIAL NERVE IDENTIFICATION

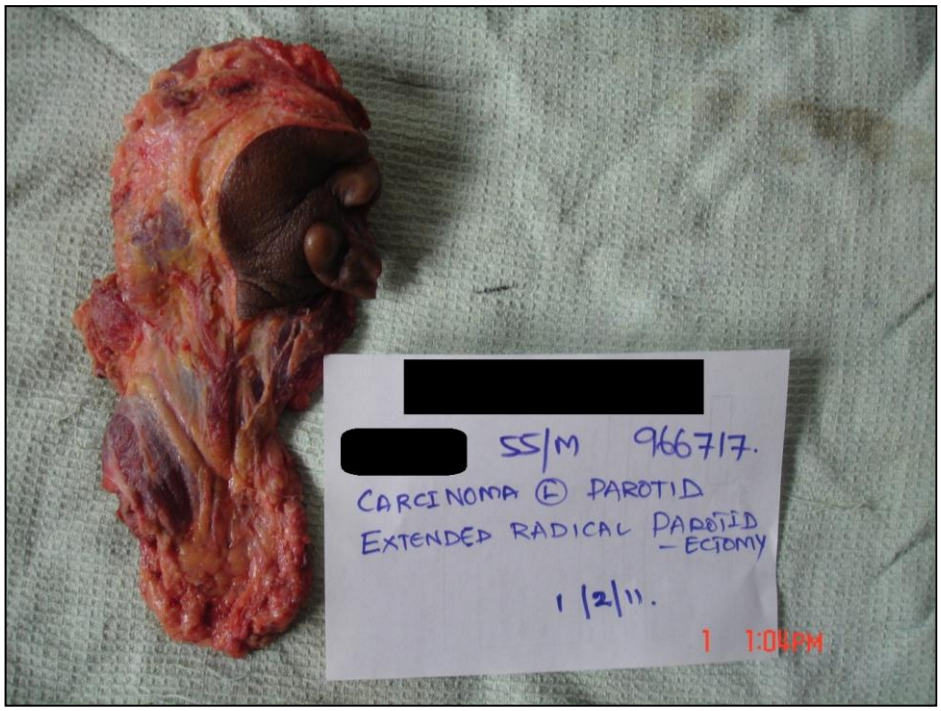


Fig. u) SPECIMEN OF EXTENDED RADICAL PAROTIDECTOMY

4. NECK DISSECTION:

In patients which detectable neck nodes, high grade tumors and infiltrative tumors it is commonly performed. The decision to employ a radical, modified radical or functional neck dissection is made based on the grade of tumor and nodal involvement.

SURGICAL COMPLICATIONS:

Complications which occur due to parotidectomy are related to the meticulous dissection, the lesion and anatomy identification. The most commonly seen complication is facial nerve involvement, which could be temporary or permanent. Other complications such as Frey's syndrome though inferred from previous studies found to be common, it is now seen that the incidence is actually much less than expected. A study[18] classified the complications based on the time of occurrence, into intra-operative, early and late complications. The following possible complications can be seen:

1. Facial nerve palsy: It may be transient of permanent. If the nerve transection is identified intra-operatively primary repair may be done either as a tension free anastomosis or interposition nerve grafts. An incidence ranging from 17% to 100% can occur as transient facial nerve paralysis depending upon the extent of resection and tumour location. [10]. Permanent paralysis has been seen fortunately in less than 5% of cases. [10].

2. Gustatory sweating (Frey's syndrome) : It occurs when there is cross-innervation of parasympathetic and sympathetic fibres which supply the parotid. On chewing food there is sweating, flushing and skin warmth. Symptomatic treatment alone is adequate in a majority of patients. Though there it has been extensively been written about, its general incidence is low, but varies with centres. Some have reported as high as 50%[18]. Conservative management is usually advocated with application of topical anti-perspirants but surgeries such as superficial temporal artery fascial flap positioning are also done in severe cases. Newer modalities to treat this condition include botulinum toxin (BTX) injection.

3. Salivary fistula: Also called sialocele it is usually self-limiting condition seen in 1%–15 % of parotidectomies[10]. Its attributed more to gland disruption than injury to Stenson's duct. Conservative management with anti-cholinergics may be done, other than which at times completion parotidectomy and low dose radiation may be employed. Staffieri et al. first proposed, in 1999, BTX in the treatment of salivary fistula and sialoceles after conservative treatment failure [19].

4. Seroma

5. Flap necrosis- it has been noted to occur at the distal tip of the posterior auricular flap.

6. Keloid formation
7. Cosmetic deformity
8. Hemorrhage
9. Wound infection
10. Hypoaesthesia over Greater auricular nerve distribution

Fig.v) FLAP NECROSIS SEEN ON 6TH POST-OPERATIVE DAY

RADIOTHERAPY:

Indications:

1. Highly malignant, aggressive tumor
2. Invasion of tissues adjacent to parotid capsule
3. Regional lymph nodal metastasis
4. Deep lobe cancers

5. Recurrent tumors

6. Facial nerve infiltration by tumor.

The minimal treatment volumes for the parotid lesions are the parotid bed and upper neck nodes. Tumour dose to the primary area is around 60 to 65 Gy in a period over 7 weeks. Higher dosage is used for microscopic positive margins or gross disease.

Complications:

1. Xerostomia 2. Tismus 3. Otitis media 4. Osteoradionecrosis 5.hair loss

CHEMOTHERAPY:

Cisplatin, paclitaxel, vinorelbine, epirubicin and mitoxantrone have had good response in around 10%–20% of studies. But response is still limited and several trials are ongoing. Trials are also underway for targeted therapy.

AIM OF THE STUDY

1. To study the incidence of various of parotid swellings in our institution.

2. To discuss accuracy of FNAC in comparison to the histo-pathological reports in our institution.

3. To study the various surgical modalities of treatment of parotid swellings applied in our institution.

4. To discuss the post-operative complications in our institution.

5. To compare findings of the above study with world statistics.

MATERIALS AND METHODS

The cohort study which included 45 patients was conducted at Kilpauk medical college hospital and Government Royapettah Hospital from September 2010 to October 2012. Data was collected from the patients after obtaining an informed consent. The study group consisted of 19 males and 26 female patients . The age group ranged from 16 years to 77 years. FNAC was performed in all patients. A total of 7 non-neoplastic, 22 neoplastic and 16 malignant lesions were identified. Forty one patients were operated on and histopathology of specimens was done in 41 cases, which included 4 cases where only biopsy was performed. Postoperative radiotherapy was given in 10 cases while palliative radiotherapy was provided in 4 cases. One case underwent chemotherapy.

Inclusion criteria :

Patients with parotid swellings neoplastic and non-neoplastic

Age of 12years and above.

Exclusion criteria:

Patients with parotid lesions due to systemic or metabolic illness.

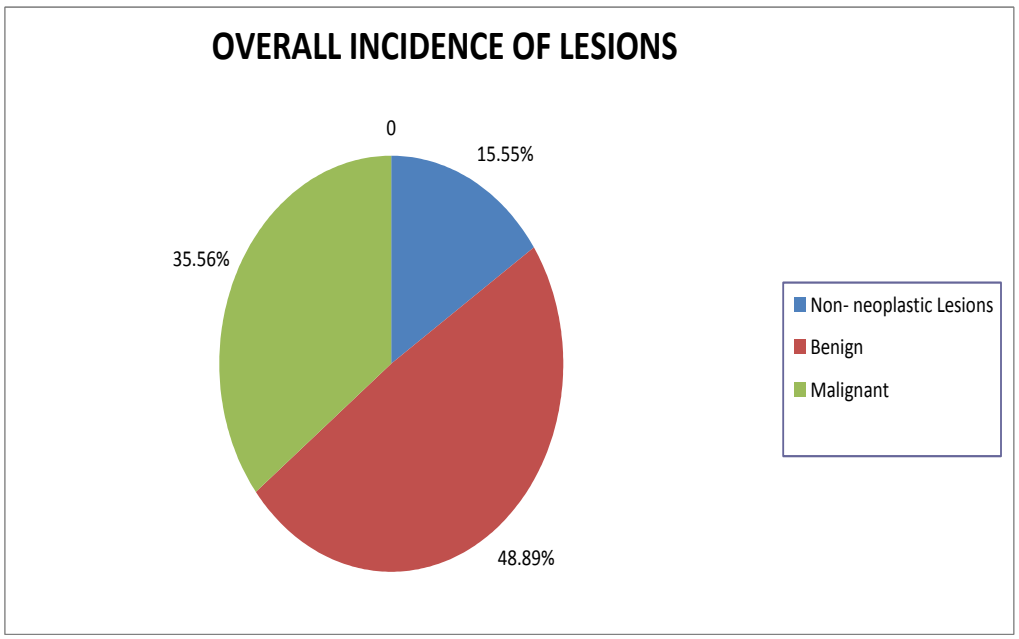
Age less than 12 years

OBSERVATION AND ANALYSIS

The observation of the study of 45 parotid lesions yielded the following results:

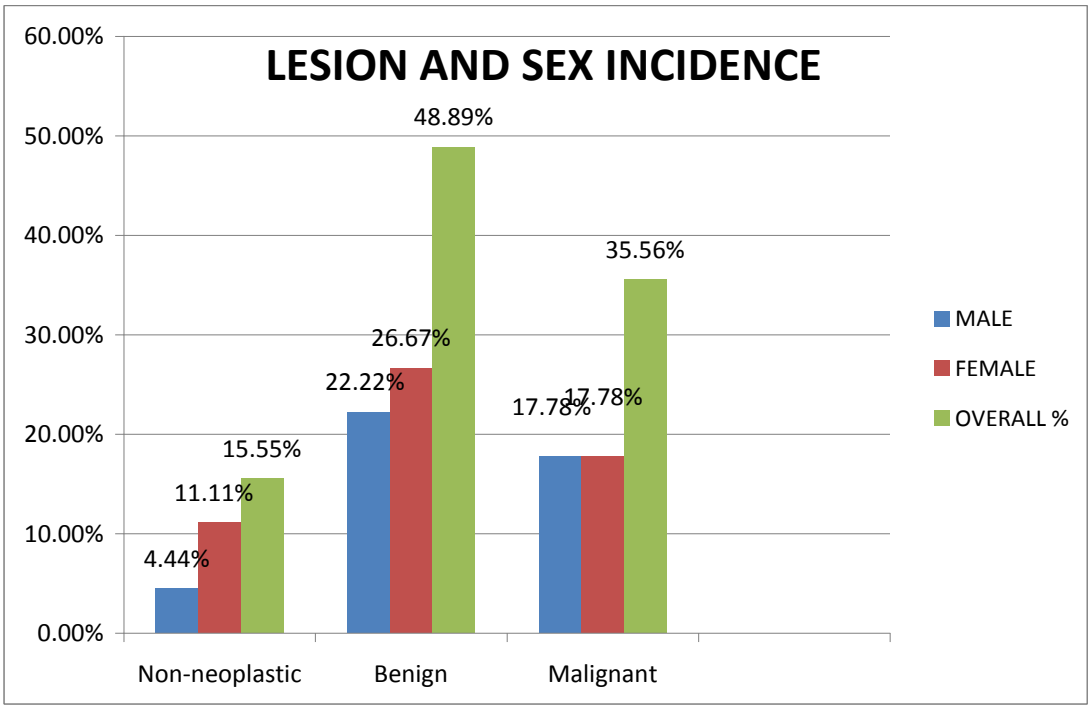
1. OVERALL INCIDENCE

LESION	TOTAL NUMBER OF CASES	PERCENTAGE
Non- neoplastic	7	15.55%
Benign	22	48.89%
Malignant	16	35.56%



2. SEX INCIDENCE DISTRIBUTION THE FOLLOWING RESULTS WERE NOTED.

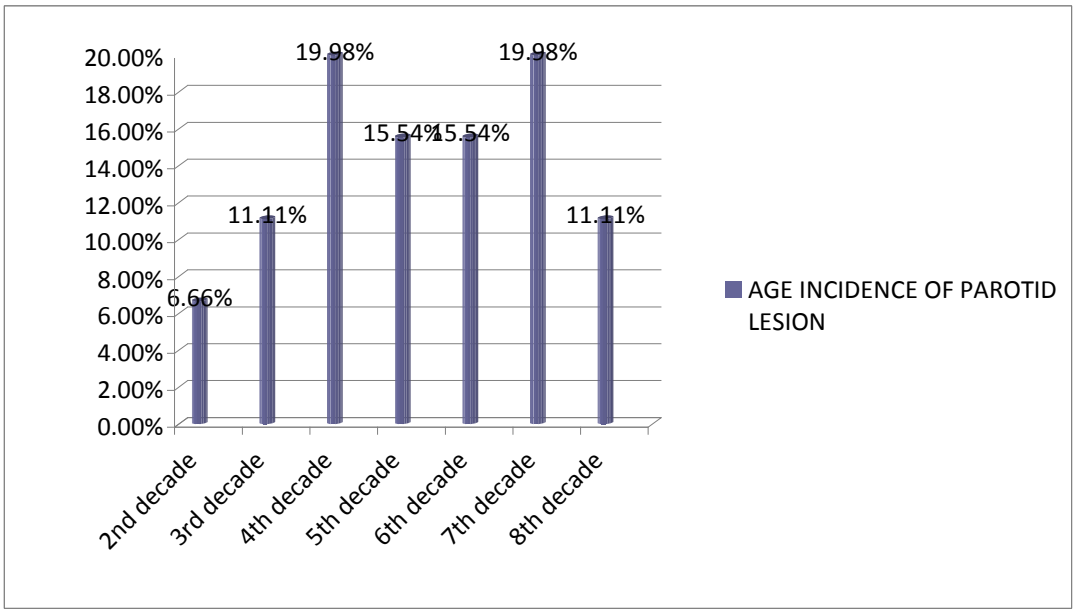
	Male	**Female**	**Total**
Non-neoplastic	4.44%	11.11%	15.55%
Benign tumor	22.22%	26.67%	48.89%%
Malignant tumour	17.78%	17.78%	35.56%



3. AGE INCIDENCE:

Age group	No. of cases	Percentage
10-16	3	6.66%
20-29	5	11.11%
30-39	9	19.98%
40-49	7	15.54%
50-59	7	15.54%
60-69	9	19.98%
70-79	5	11.11%

AGE INCIDENCE OF PAROTID LESION



4. FNAC

FNAC has been discussed subsequently under the respective lesions.

5. MANAGEMENT

A. SURGICAL

Superficial parotidectomy was the most commonly performed surgery. Total conservative parotidectomy was the second most common.

TYPE OF SURGERY	**NO. OF CASES**	**PERCENTAGE**
SUPERFICIAL PAROTIDECTOMY	25	55.55%
TOTAL PAROTIDECTOMY	6	13.33%
RADICAL PAROTIDECTOMY	1	2.22%
EXTENDED RADICAL PAROTIDECTOMY	3	6.66%
INCISION & DRAINAGE	3	6.66%
EXCISION	1	2.22%
COMPLETION PAROTIDECTOMY	2	4.44%

B. NON-SURGICAL MODALITIES

MODALITY	**RADIOTHERAPY**		**CHEMOTHERAPY**
	POST OPERATIVE	**PALLIATIVE**	
NO. OF CASES	10	4	1

6. POST-OPERATIVE COMPLICATIONS:

Complication	Percentage
Facial nerve palsy	20%
Seroma	20%
Flap Necrosis	4.44%
Fistula	4.44%
Others	2.22%

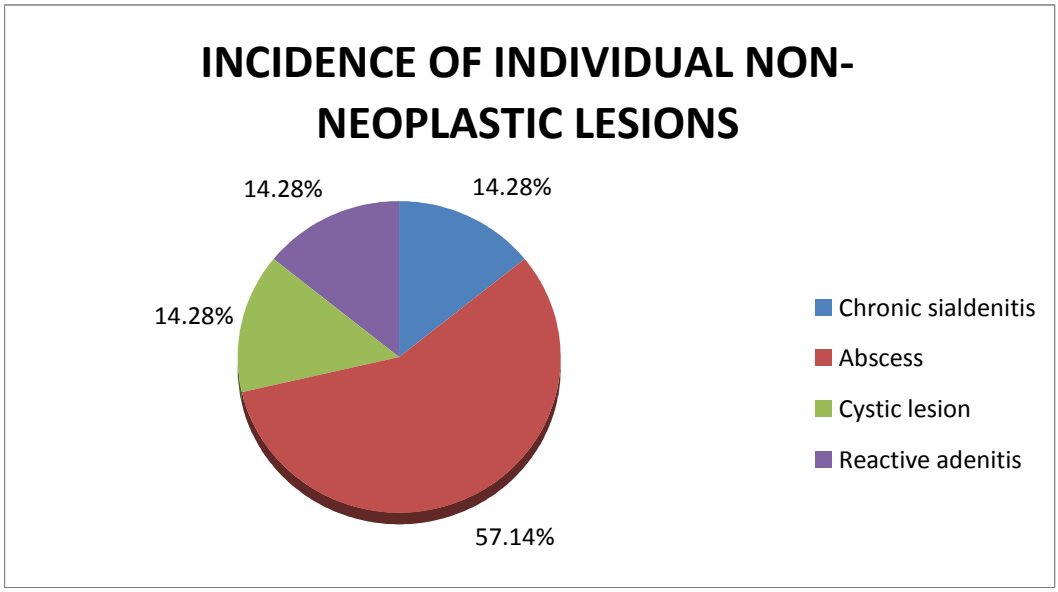
Among the other complications it was seen that one patient had vocal cord paralysis.

Analysis of individual groups of lesions yielded the following results:

1. NON- NEOPLASTIC LESION:

The study of the lesions revealed the following results

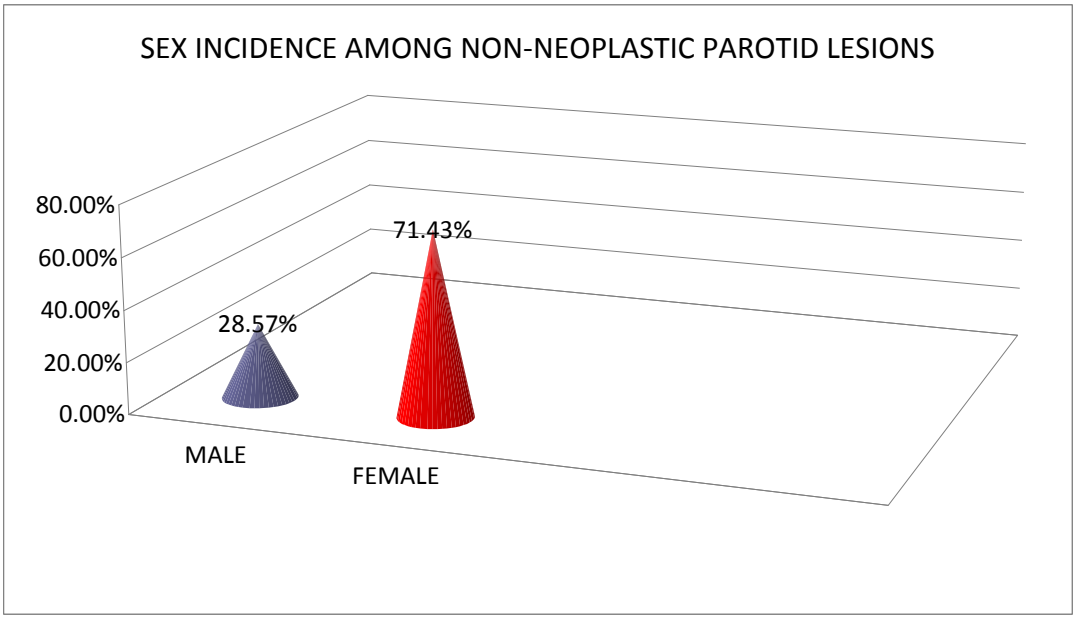
LESION	MALE	FEMALE
Chronic Sialadenitis	-	1
Abscess	1	3
Cystic lesion	1	-
Reactive adenitis	-	1



Abscess formed a majority of the non-neoplastic group with 4 out of 7 cases.

2. SEX INCIDENCE:

SEX	NO. OF CASES	PERCENTAGE	OVERALL PERCENTAGE(ALL LESIONS)
MALE	2	28.57%	4.44%
FEMALE	5	71.43%	11.11%



Women were more affected by non-neoplastic parotid lesions than men having 71.43% of the lesions.

3. FNAC

In the cytological analysis it was noted that, although there was a higher rate of lesions which were positive, the true positives were lesser.

Lesion	Positive FNAC	Accuracy
Chronic sialadenitis	2	50%
Abscess	5	100%
Cystic lesion	2	50%
Reactive adenitis	1	100%

On further evaluvation of 3 lesions, they turned out to be Non-Hodgkin's lymphoma, mucoepidermoid carcinoma and pleomorphic adenoma. Hence the overall accuracy of FNAC is around 70%.

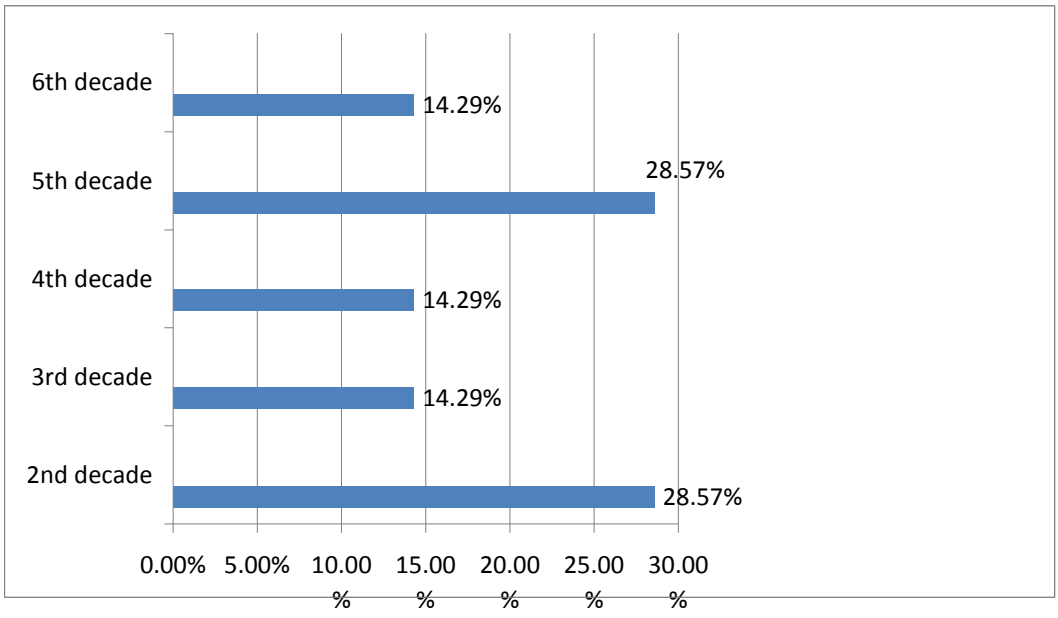
In the data set of our study, it was found to be highly sensitive and highly specific.

Positive predictive value of the 77% was found with this data set.

4. Age incidence:

Age group	No. of patients	Percentage
10-19	2	28.57%
20-29	1	14.29%
30-39	1	14.29%
40-49	2	28.57%
50-59	1	14.29%

AGE INCIDENCE FOR NON NEOPLASTIC LESION

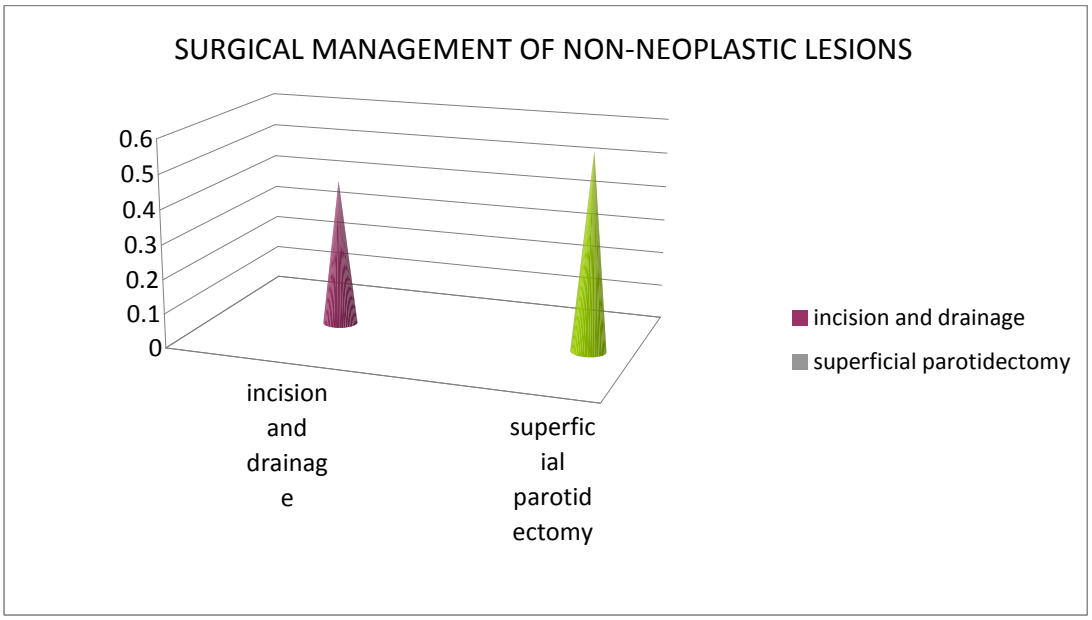


5. Presentation:

Presentation	No. of patients	Percentage
Painful swelling	1	14.29%
Painless swelling	6	85.71
Discharge	Nil	-
Facial nerve palsy	Nil	-
Node enlargement	Nil	-

6. Treatment:

TREATMENT	PERCENTAGE	OVERALL PERCENTAGE
Incision and drainage	42.86%	6.66%
Superficial parotidectomy	57.14%	8.88%



7. Complications:

Significant complications noted in this was seen only in one case where seroma and parotid fistula developed. These resolved on its own with time and conservative medical management.

7. NEOPLASTIC LESIONS:

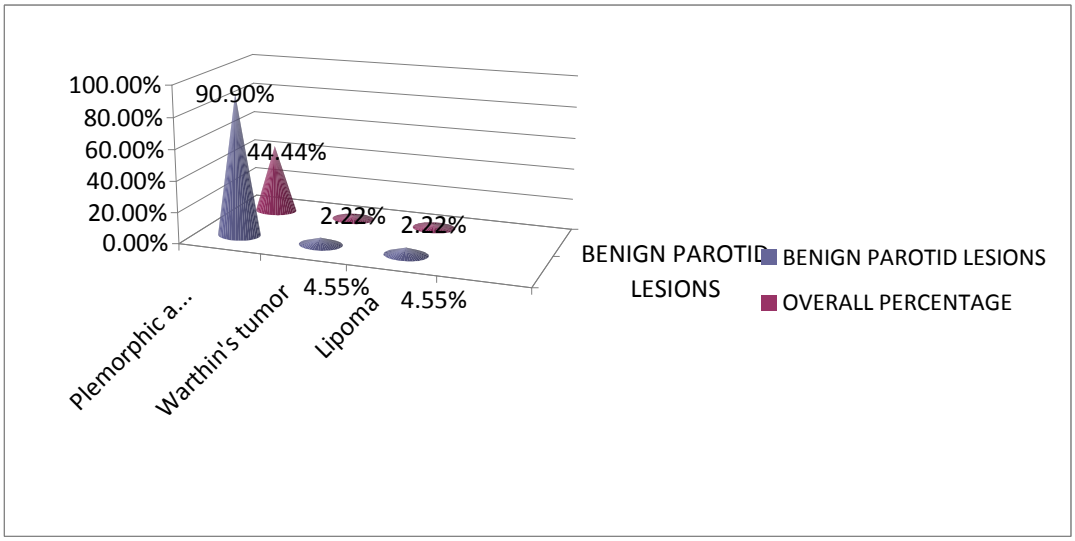
A. BENIGN LESIONS:

It was seen that among the benign lesions, pleomorphic adenoma was a dominant lesion. A total of 22 benign neoplasms were present in the study which comprised of 48.88% of all lesions in the study.

1. Lesion incidence:

Lesion	No. of cases	Percentage	Overall percentage
Pleomorphic adenoma	20	90.9%	44.44%
Warthin's tumor	1	4.55%	2.22%
Lipoma	1	4.55%	2.22%

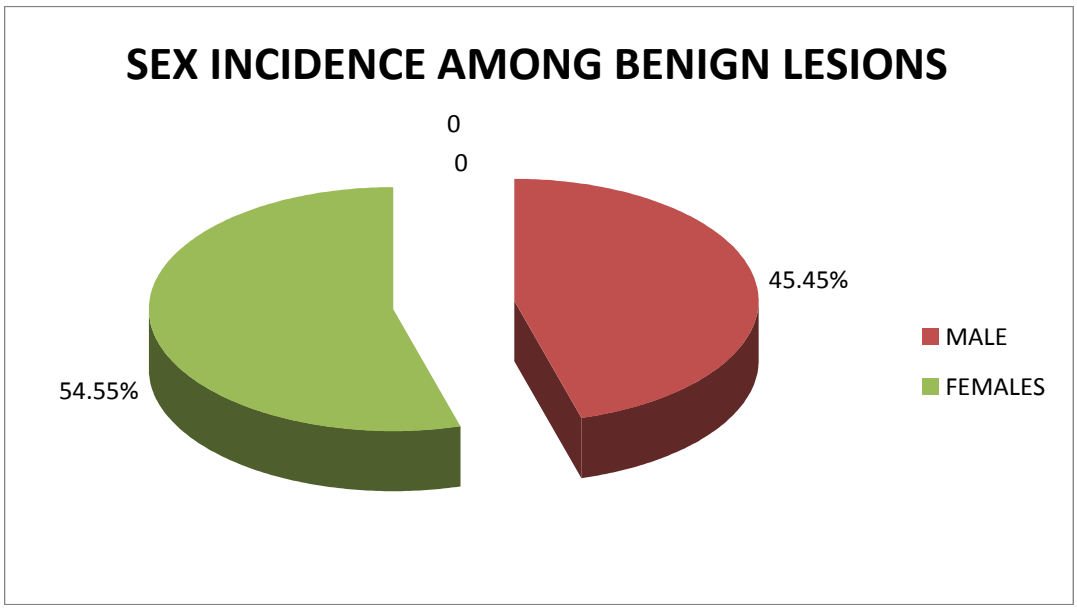
INCIDENCES OF INDIVIDUAL BENIGN TUMOURS



2. Sex incidence:

Sex	No. of cases	Percentage	Overall percentage
Male	10	45.45%	22.22%
Female	12	54.55%	26.66%

In this category it was seen that though the incidence was marginally more among females, males too were maximum affected by benign lesions.

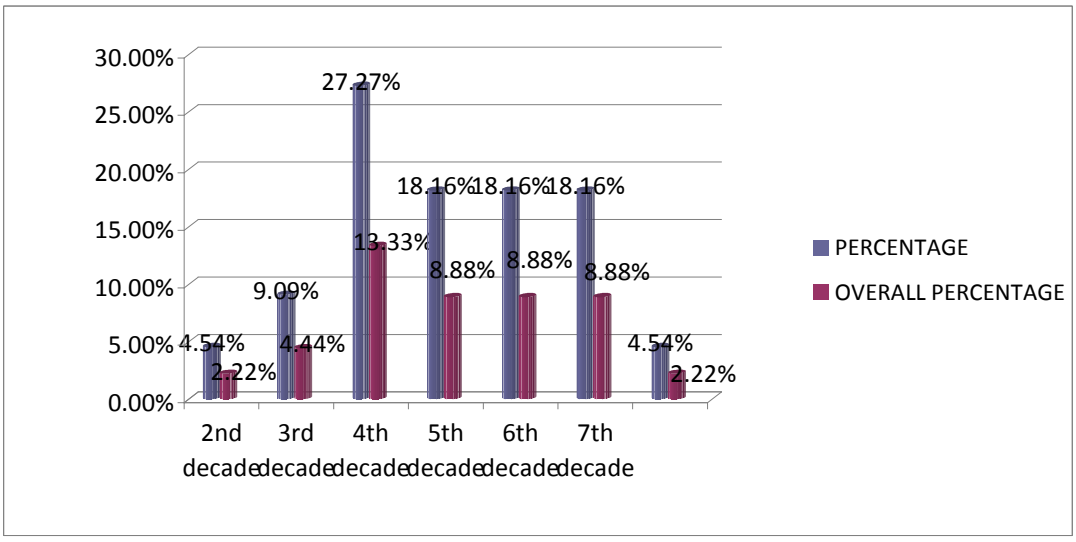


3. AGE INCIDENCE:

AGE GROUP(years)	NO. OF PATIENTS	PERCENTAGE %	OVERALL PERCENTAGE (%)
10-19	1	4.54%	2.22%
20-29	2	9.09%	4.44%
30-39	6	27.27%	13.33%
40-49	4	18.16%	8.88%
50-59	4	18.16%	8.88%
60-69	4	18.16%	8.88%
70-79	1	4.54%	2.22%

Therefore the age group most susceptible to benign tumours is in the 4th decade. The youngest patient seen was a 16 year old male with a lipoma.

AGE INCIDENCE FOR BENIGN TUMOURS



4. PRESENTATION:

Presentation	No. of cases	Percentage
Swelling without pain	21	46.66%
Swelling with pain	1	2.22%
Facial nerve palsy	Nil	
Discharge	Nil	
Deep lobe involvement	Nil	
Recurrence	3	6.66%

5. FNAC

Lesion	Total no. detected
Pleomorphic adenoma	19
Warthin's tumor	1
Lipoma	1

In one patient it the FNAC showed a cystic aspirate when it was actually a pleomorphic adenoma, therefore making the total number of pleomorphic adenoma to 20.

Sensitivity: = 100%

Specificity = 86.67%

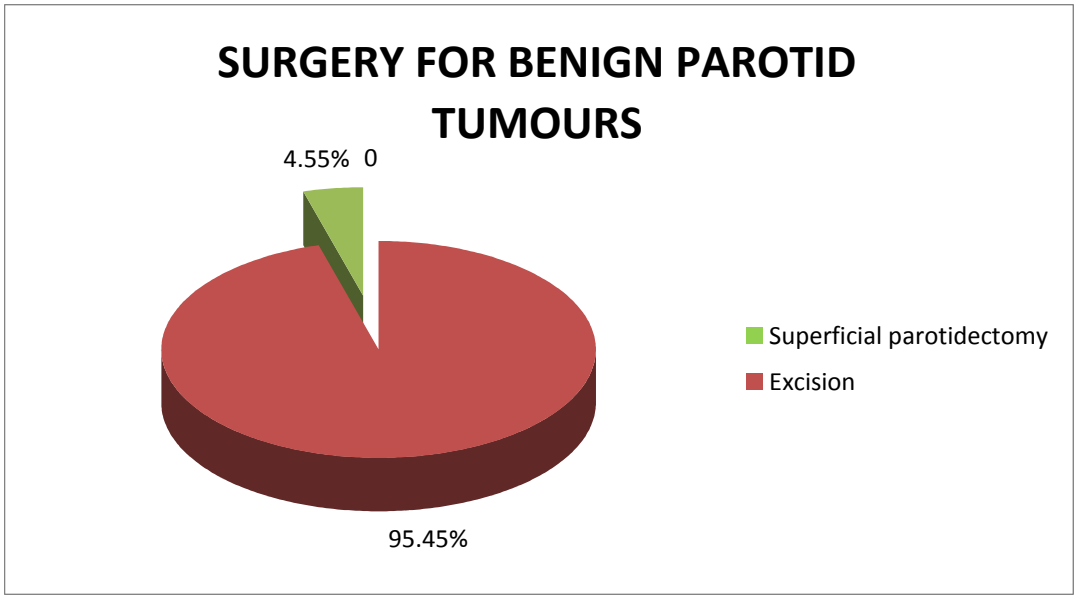
Positive predictive value = 93.75%

Negative predictive value = 100%

6. TREATMENT:

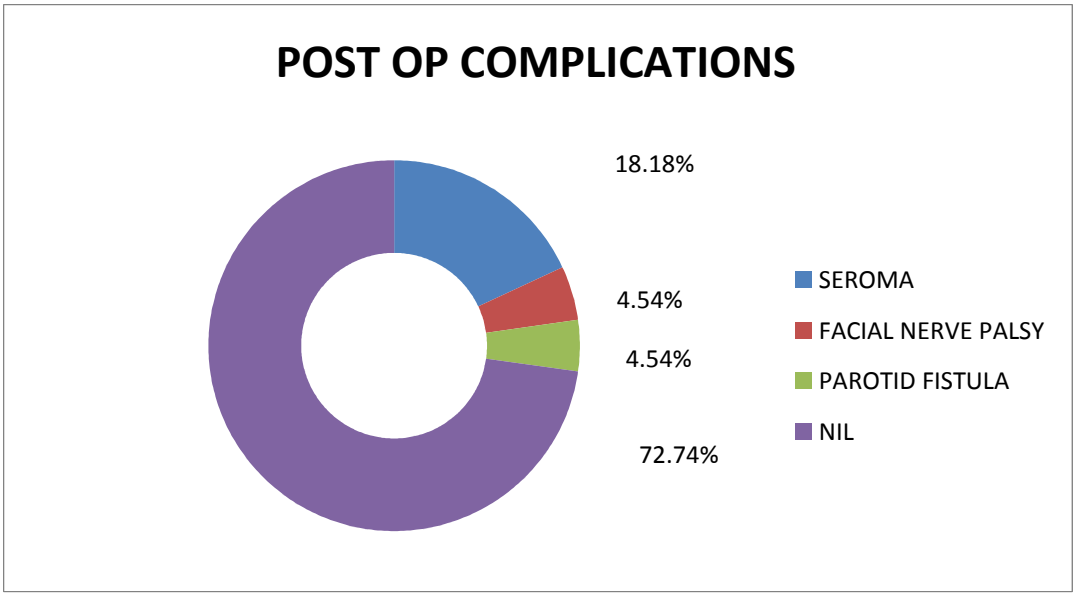
Surgery was the mainstay treatment of the benign lesions. Most underwent superficial parotidectomy barring the case of lipoma who underwent excision of the lesion alone.

Surgery	No. of cases	Percentage overall	Percentage
Superficial parotidectomy	21	46.67	95.45
Excision	1	2.22	4.55



7. POST- OPERATIVE COMPLICATIONS:

Complication	No. of patients	Percentage of cases
Seroma	4	18.18%
Facial nerve palsy	1	4.54%
Parotid fistula	1	4.54%

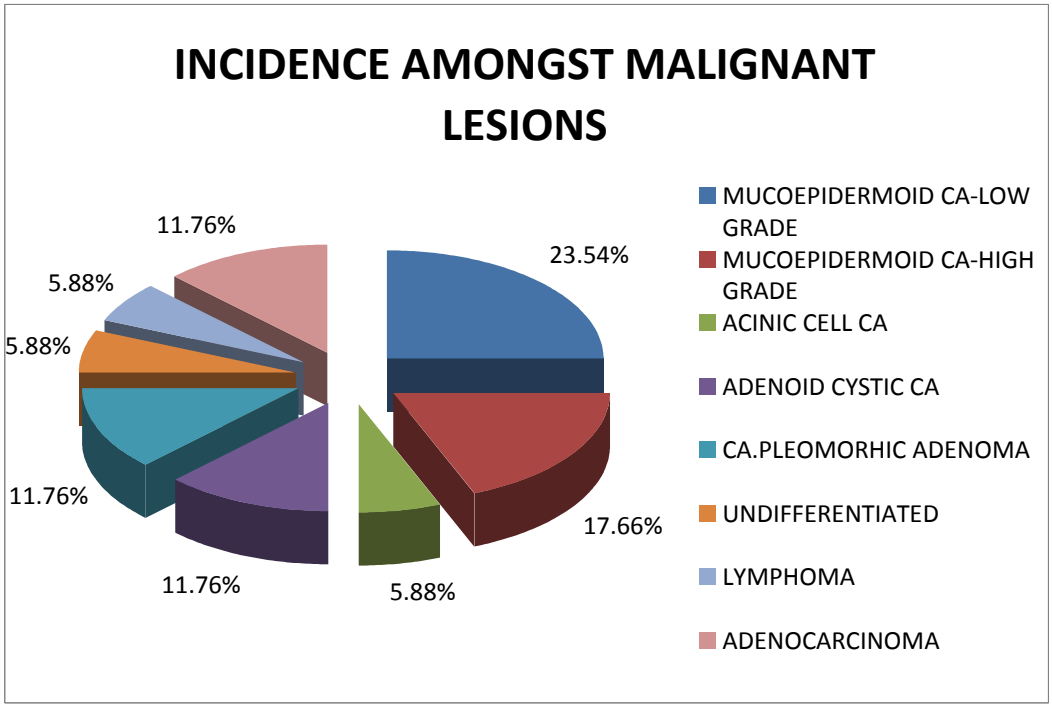


MALIGNANT LESIONS:

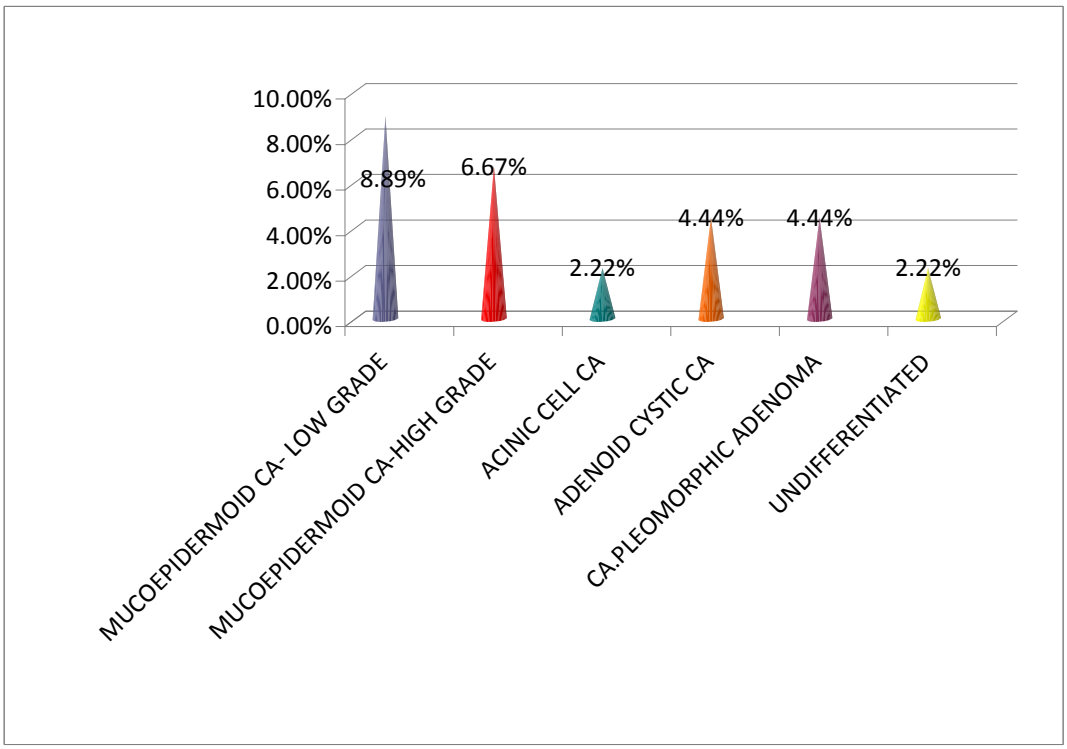
A total of 17 cases of malignant parotid tumors were there in our study, of which the predominant type seen was mucoepidermoid carcinoma comprising of 7 cases.

1. Incidence

Lesion	**No. of cases**	**Percentage**	**OVERALL % OF INCIDENCE**
Mucoepidermoid carcinoma- low grade	4	23.54%	8.89%
Mucoepidermoid carcinoma- high grade	3	17.66%	6.67%
Acinic cell carcinoma	1	5.88%	2.22%
Adenoid cystic carcinoma	2	11.76%	4.44%
Carcinoma ex pleomorphic adenoma	2	11.76%	4.44%
Undifferentiated	1	5.88%	2.22%
Lymphoma(NHL)	1	5.88%	2.22%
Adenocarcinoma	2	11.76%	4.44%



OVERALL INCIDENCE:

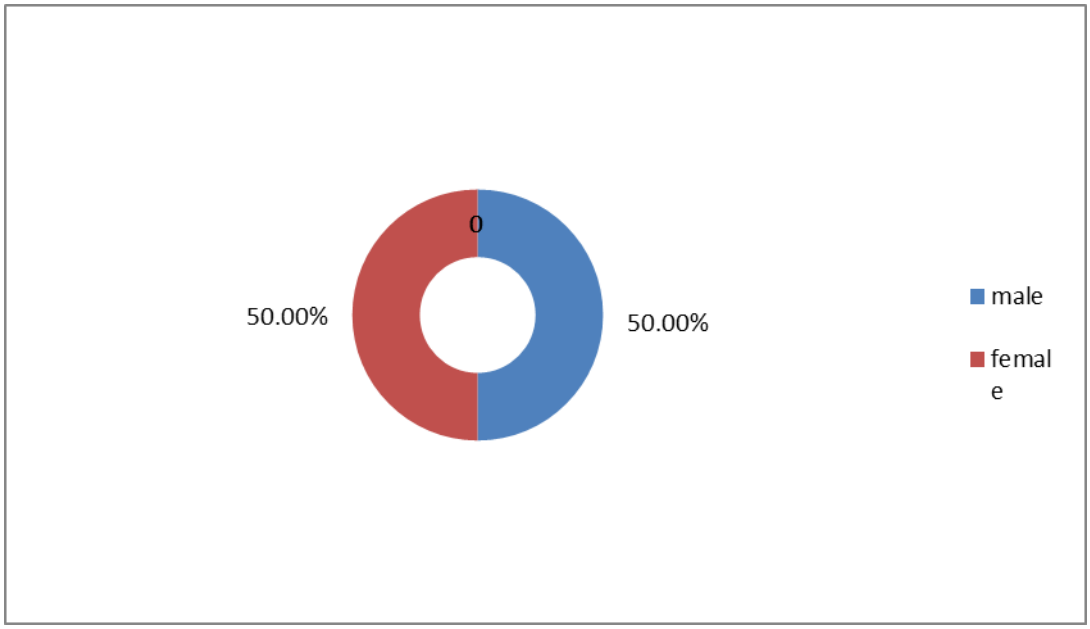


1. SEX INCIDENCE:

Sex	No. of cases	Percentage	Overall percentage
Male	8	50%	17.78%
Female	8	50%	17.78%

It was seen that the malignancies of the parotid affected men and women with equal incidence.

SEX INCIDENCE

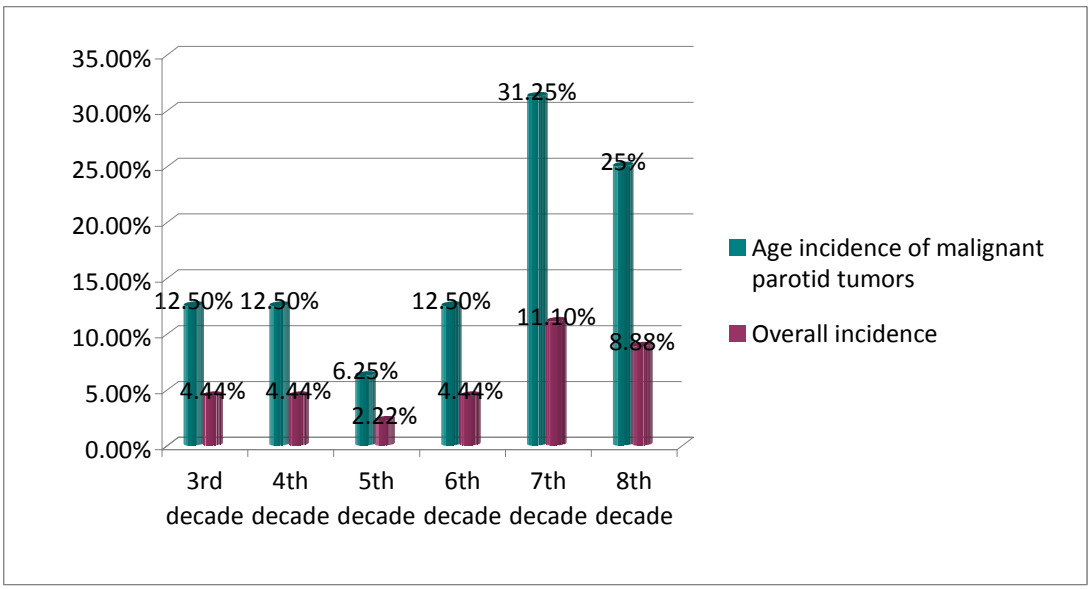


1. AGE INCIDENCE:

AGE(years)	NO. OF CASES	PERCENTAGE	OVERALL PERCENTAGE
10-19	NIL		
20-29	2	12.5%	4.44%
30-39	2	12.5%	4.44%
40-49	1	6.25%	2.22%
50-59	2	12.5%	4.44%
60-69	5	31.25%	11.1%
70-79	4	25%	8.88%

The maximum incidence of malignancies was noted too be in the 7^{th} decade seen in 31.25% of malignancies closely followed by the 8^{th} decade affected in 25%.

AGE INCIDENCE FOR MALIGNANT TUMOURS

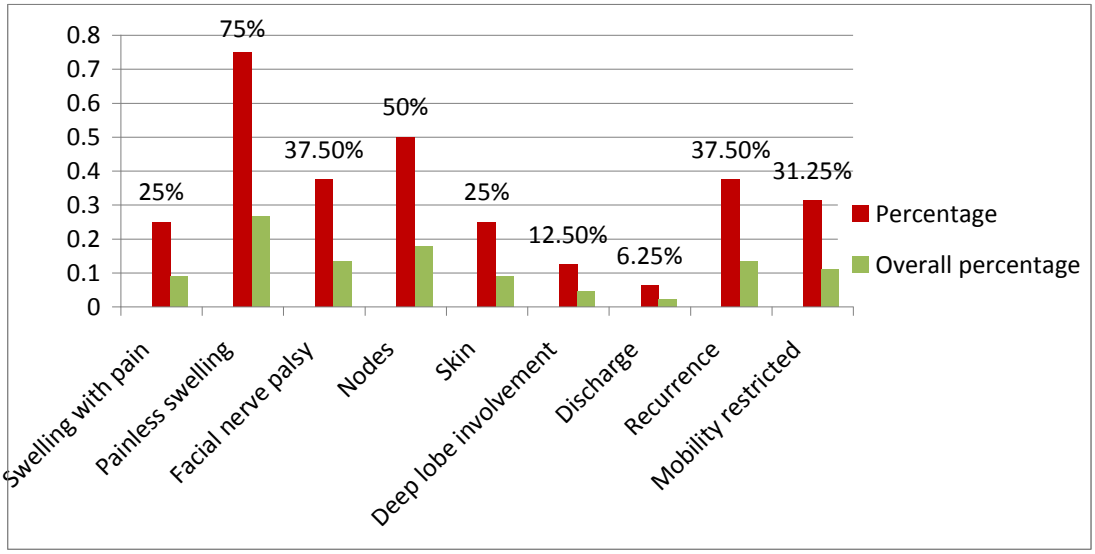


4. Presentation

Presentation	No. of cases	Percentage	Overall percentage
Swelling with pain	4	25%	8.88%
Painless swelling	12	75%	26.67%
Facial nerve palsy	6	37.5%	13.33%
Nodes	8	50%	17.78%
Skin	4	25%	8.89%
Deep lobe involvement	2	12.5%	4.44%
Discharge	1	6.25	2.22%
Recurrence	6	37.5%	13.33%
Mobility restricted	5	31.25%	11.11%

It was noted that half the cases of malignant parotid tumours presented with enlarged cervical nodes and a significant number of cases (37.5%) had associated facial nerve palsy.

AGE INCIDENCE FOR MALIGNANT PAROTID TUMOURS



FNAC

LESION	Total no. Of positives
Mucoepidermoid carcinoma	6
Acinic cell carcinoma	3
Adenoid cystic carcinoma	nil
Adenocarcinoma	nil
Carcinoma pleomorphic adenoma	2
Malignant cells	3

With FNAC it was seen that 14 malignant tumors were detected. The final histopathology report showed that a total of 16 malignant parotid lesions were present.

It was seen that there were several errors in the detection of malignant lesions, not only regarding the type of lesion that was detected but in fact many were concluding a wrong result

Sensitivity	=	87.5%
Specificity	=	100%
Positive predictive value	=	100%
Negative predictive value	=	93.55%

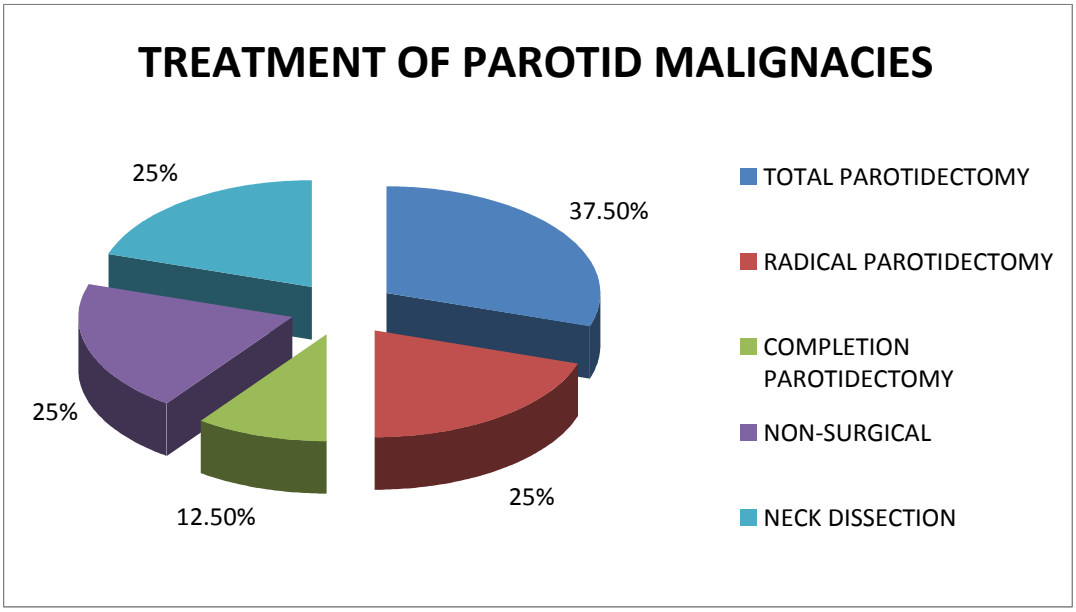
2. TREATMENT MODALITIES:

Surgical modality was the mainstay treatment for most cases and the most commonly performed surgery was total parotidectomy, it comprised around 37.5%. Also neck dissection was performed in 31.5% of the cases.

Surgery	No. of cases	Percentage	Overall percentage
Total conservative parotidectomy	6	37.5%	13.33%
Radical parotidectomy(including extended radical parotidectomy)	4	25%	8.89%
Completion parotidectomy	2	12.5%	4.44%
Neck dissection	5	31.25%	11.11%
Non-surgical management	4	25%	8.89%

Other modalities

Radiotherapy was used in almost all cases, either in the form of palliation or post-operatively as an adjuvant. Adjuvant radiotherapy was given in 62.5% of the cases of malignancies and as palliation in 31.25% of cases. Chemotherapy was given as an adjuvant in one case where the diagnosis was Non-Hodgkin's lymphoma (CHOP Regimen) and as palliation 1 case.

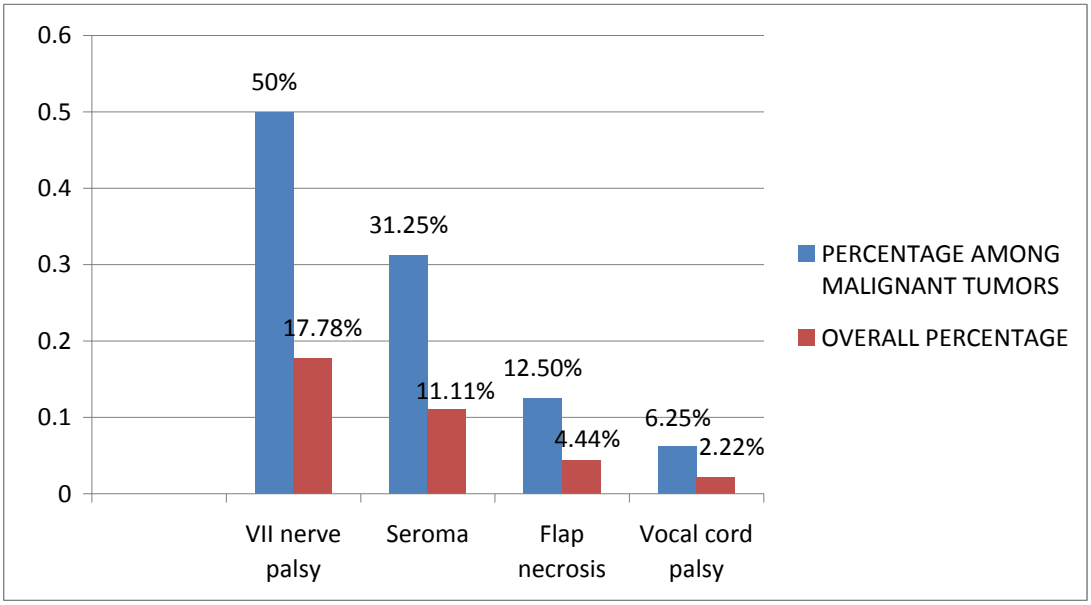


3. POST-OPERATIVE COMPLICATIONS:

The most common post-operative complication noted in this group of patients was facial palsy due to injury to the facial nerve, seen in 50% of the cases. Also seroma formation was noted in 31.25% of the patients who underwent surgery. One patient had vocal cord palsy, due to the extensive dissection and resection of the infiltrative tumour.

COMPLICATION	NO. OF CASES	PERCENTAGE	OVERALL PERCENTAGE
VII nerve palsy	8	50%	17.78%
Seroma	5	31.25%	11.11%
Flap necrosis	2	12.5%	4.44%
Vocal cord palsy	1	6.25%	2.22%

POST-OPERATIVE COMPLICATIONS FOR MALIGNANT TUMOURS

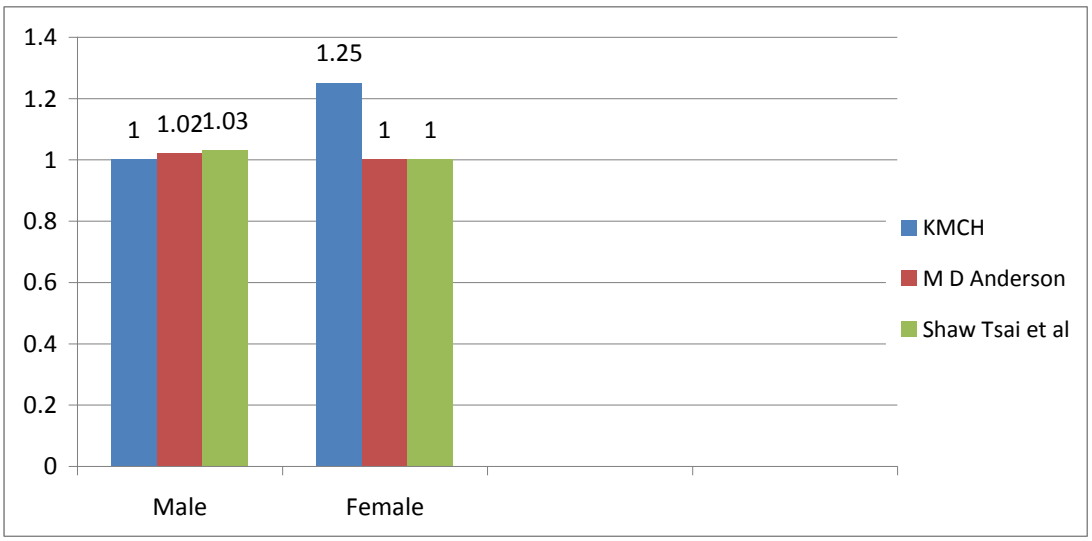


DISCUSSION

The comparative analysis of the study was made with other published studies and the following results were obtained.

1. COMPARISON OF SEX INCIDENCE BETWEEN OUR INSTITUTION AND VARIOUS CENTRES:

Institution	**KMC**	**M D Anderson cancer center[8]**	**Shaw Tsai et al[6]**
Male:female	1:1.25	1.02: 1	1.03:1
Male	20	77	53
Female	25	75	55

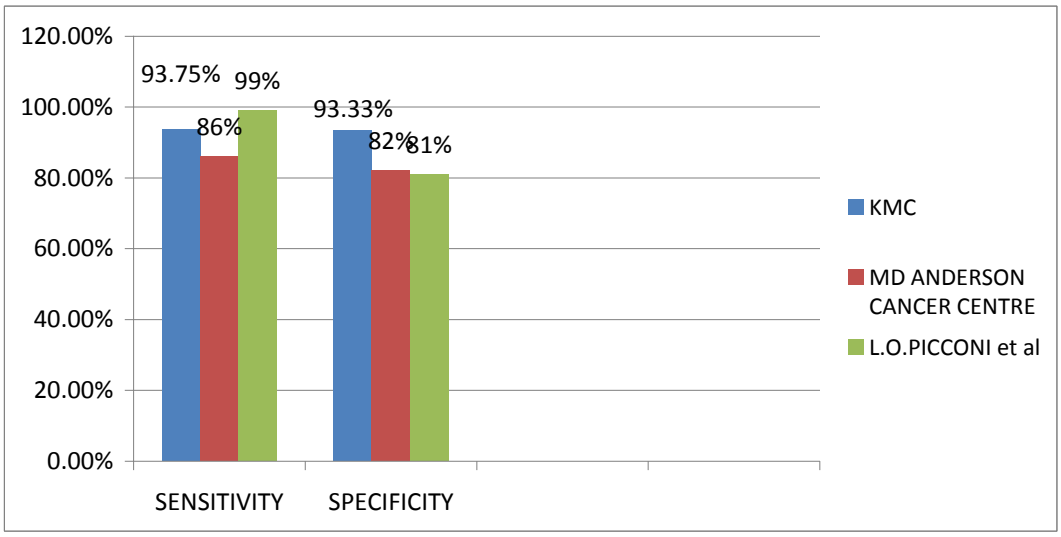


Our centre showed a slightly higher incidence among females in comparison to other centres.

1. FNAC COMPARISON BETWEEN OUR INSTITUTION AND VARIOUS CENTRES

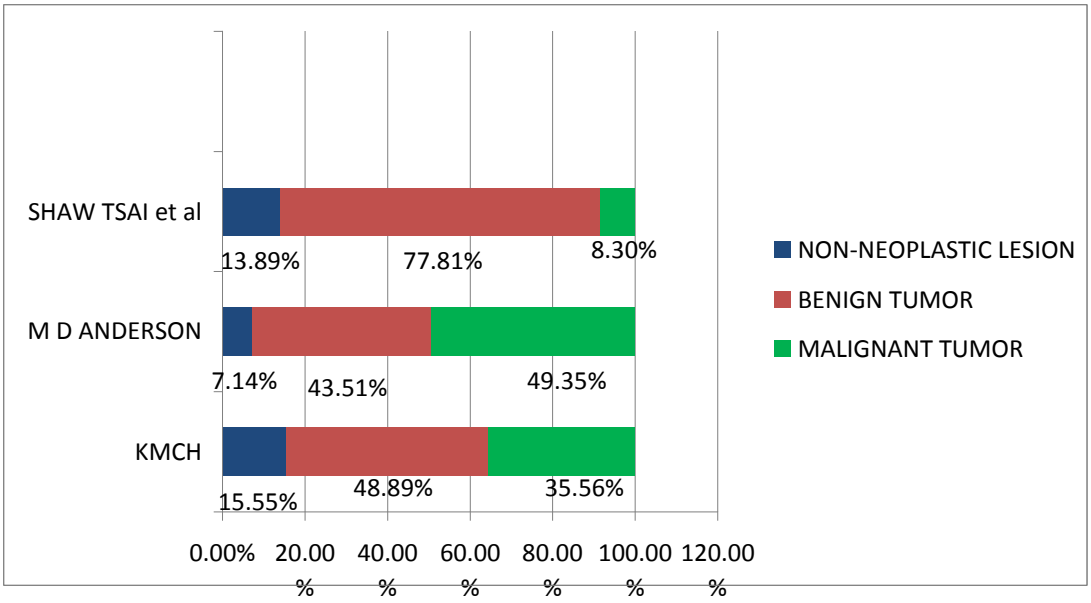
CENTRE	KMC	M D ANDERSON CANCER CENTRE[8]	Italy, picconi et al[14]
SPECIFICITY	93.75%	86%	99%
SENSITIVITY	93.33%	82%	81%
PPV	96.87%	85%	93%
NPV	96.77%	86%	98%

FNAC COMPARISON AMONG VARIOUS CENTRES



2. Incidence of various lesions among institutes:

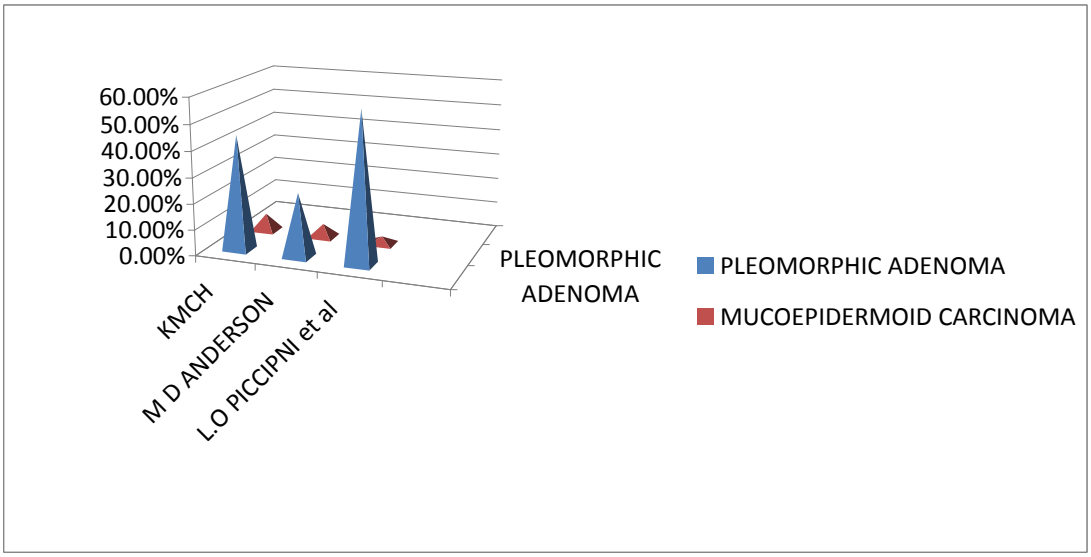
Centre	NON-NEOPLASTIC LESIONS	BENIGN TUMORS	MALIGNANT TUMORS
KMCH	15.55%	48.89%	35.56%
M D ANDERSON[8]	7.14%	43.51%	49.35%
Shaw Tsai et al[6]	13.89%	77.81%	8.3%



It was seen that among all the centers results for most common benign and malignant lesions were corresponding with each other, with pleomorphic adenoma and mucoepidermoid carcinoma respectively.

LESION	BENIGN LESION	MALIGNANT TUMOR
KMCH	PLEOMORPHIC ADENOMA (44.44%)	MUCOEPIDERMOID CARCINOMA (6.67%)
MD ANDERSON[8]	PLEOMORPHIC ADENOMA (24.68%)	MUCOEPIDERMOID CARCINOMA (5.19%)
L.O. Piccioni et al [14]	PLEOMORPHIC ADENOMA(57.85%)	MUCOEPIDERMOID CARCINOMA(2.85%)

INCIDENCE OF PLEOMORPHIC ADENOMA AND MUCO-EPIDERMOID CARCINOMA AMONG THE INSTITUTES

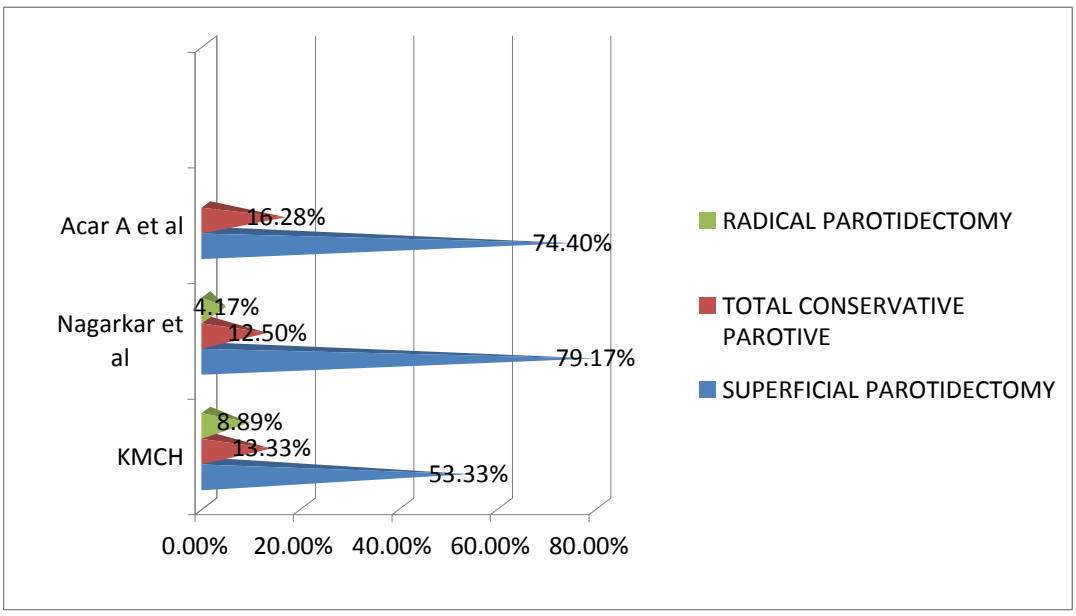


3. SURGICAL MANAGEMENT:

	Superficial parotidectomy	Total conservative parotidectomy	Total parotidetomy with facial nerve resection	Radical parotidectomy	Neck dissection	Others
KMCH	53.33%	13.33%		8.89%	11.11%	13.33%
Nagarkar et al[24]	79.17%	12.5%		4.17%	4.17%	4.16%
Acar A, *et al*[25]	74.4%	16.28%	6.98%	-	13.95%	2.32%

In our study it was found that the other surgeries performed were completion parotidectomy, incision drainage and excision biopsies which corresponded with other studies.

SURGICAL MANAGEMENT AT VARIOUS CENTRES



OTHER TREATMENT MODALITIES:

Radiotherapy was used as a postoperative adjuvant management in most cases of malignant tumors.

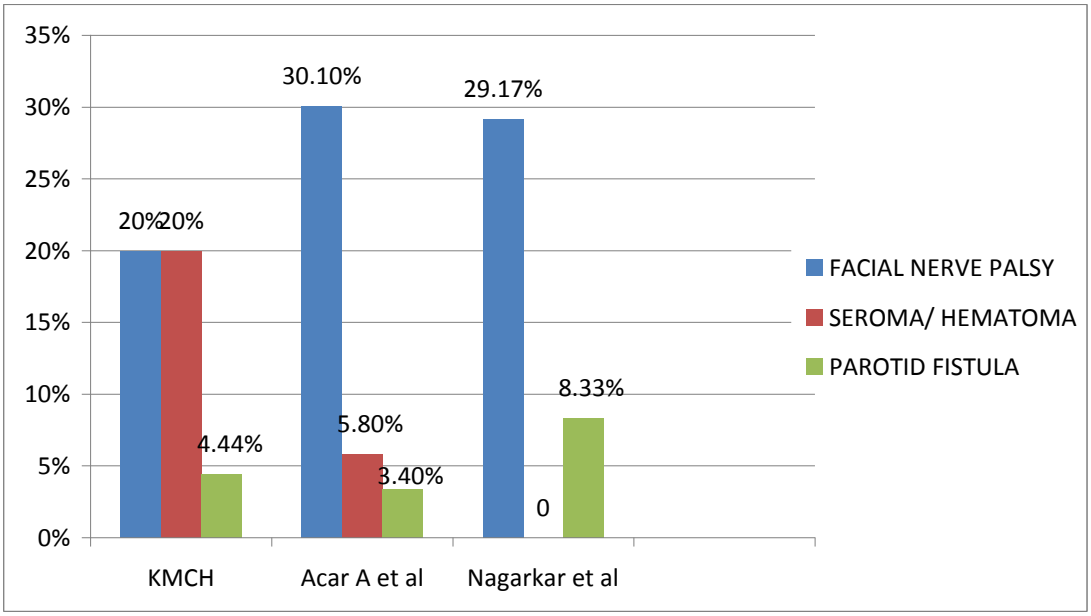
	POST OPERATIVE RADIOTHERAPY
KMCH	62.5%
Acar A *et al*[25]	84.4%
Nagarkar et al[24]	20%

4. Postoperative complications

Most common complication of surgery was noted to be facial nerve palsy, transient or permanent.

POSTOP COMPLICATION	**FACIAL NERVE PALSY**	**SEROMA/ HEMATOMA**	**FLAP NECROSIS/ INFECTION**	**PAROTID FISTULA**
KMCH	20%	20%	4.44%	4.44%
Acar A et al [25]	30.1%	5.8%	4.6%	3.4%
Nagarkar et al[24]	29.17%	-	-	8.33%

POST-OPERATIVE COMPLICATIONS AT VARIOUS CENTRES



CONCLUSION

The analysis of the data of the study conducted at our institution provided us with the following results:

1. Parotid lesions comprised of the most common salivary gland lesions in our hospital.
2. Amongst the various lesions it was noted that benign tumours were the most common and the least common were non-neoplastic disorders.
3. The sex incidence showed a similar distribution among both males and females with the ratio being 1:1.25.
4. The mean age of presentation was 49 yearsand it was seen that the 4th and 7^{th} decades where the predominant age group for occurrence in case of benign and malignant tumours respectively.
5. The lesions which were predominant in the non-neoplastic, benign and malignant tumours groups where abscess, pleomorphic adenoma and mucoepidermoid carcinomas respectively. These were found to be consistent with the comparison made with world statistics.
6. FNAC correlated in a total of 39 out of 45 cases, i.e.86.67% of the cases.

The sensitivity and specificity for detection of benign tumors was found to be 93.75% and 100% respectively. In the case of malignant

tumours the sensitivity and specificity was found to be 87.5% and 100% respectively.

8. Patients presenting with facial nerve palsy was seen more amid the malignant tumors.
9. Most commonly performed surgery was superficial parotidectomy. Completion parotidectomy was performed in 2 cases and both were malignant tumors with recurrence.
10. Facial nerve palsy and seroma formation were the commonest complication noted post-operatively.
11. Radiotherapy was the most common non-surgical modality used and administered more commonly post-operatively.

BIBILOGRAPHY

1. Joseph Attie et al ; in **Tumors of major and minor salivary glands : clinical and pathologic features in** Chicago : Year Book Medical Publisher, 1981.Current problems in surgery, v. 18, no. 2.

2. Anatomy of the facial nerve branching patterns, the Marginal mandibular branch and its extra parotid Ramification in relation to the lateral palpebral line Weerapant et al, Asian Biomedicine Vol. 4 No. 4 August 2010; 603-608

3. Davis RA, Anson BJ, Budinger JM, Kurth LR. Surgical Anatomy of the facial nerve and parotid gland based upon a study of 350 cervicofacial halves. Surg Gynecol Obstet. 1956; 102:385-413.

4. Facial nerve;pattern of distribution in the parotid gland; DR. FAROOQ AHMED et al , The Professional vol:12, no:01 jan, feb, mar, 2005

5. Anatomy and physiology of the salivary glands, Grand Rounds Presentation, UTMB, Dept. Of Otolaryngology Frederick S. Rosen, MD; Byron J. Bailey, MD

6. Parotid neoplasms: diagnosis, treatment, and intraparotid Facial nerve anatomy ; Stella Chin-Shaw Tsai, M.D., Ph.D.*, Hsin-Te Hsu, M.D.; *The Journal of Laryngology & Otology*May 2002, Vol. 116, pp. 359–362

7. Lundeberg D: Nonneoplastic disorders of the parotid gland West J Med 1983 Apr; 138:589-595.

8. Fine-Needle Aspiration of 154 Parotid Masses with Histologic Correlation Ten-Year Experience at the University of Texas M. D. Anderson Cancer Center; Basim M. Al-Khafaji, M.B., Ch.B. Blake R. Nestok, M.D. Ruth L. Katz,

M.D.Cancer (Cancer Cytopathol) 1998;84:153–9. © 1998 American Cancer Society.

9. Tumours of the salivary glands; J.W. Eveson, P. Auclair, D.R. Gnepp, A.K. El-Naggar; in Head and Neck tumors, Lyon; IARC Press 2005, P 209-281

10. ACS SURGERY Principles and Practice of Surgery, 6th Edition 2007; Souba, Wiley W

11. Review of Fine-Needle Aspiration Cytology of Salivary Gland Neoplasms, With Emphasis on Differential Diagnosis *Perkins Mukunyadzi, MD;* Am J Clin Pathol 2002

12. Sonographically Guided Fine-Needle Aspiration Biopsy of Major Salivary Gland Masses: A Review of 245 Cases Hee Woo Cho1, Jinna Kim1, Junjeong Choi2, Hyun Seok Choi1, Eun Soo Kim1, Se-Heon Kim3 and Eun Chang Choi3

13. Mcwhorter, G. L.: Relations of Superficial and Deep Lobes of Parotid Gland to Duct and Facial Nerve. Anat. Rec., 12:149, 1917.

14. Fine-needle aspiration cytology in the diagnosis of parotid lesions, L.O. Piccioni, B. Fabiano, M. Gemma1, D. Sarandria, M. Bussi ACTA otorhinolaryngologica italica 2011;31:1-4

15. Zbaren P, Schar C, Hotz MA, Loosli H. Value of fine needle aspiration cytology of parotid gland masses. Laryngoscope 2001;111:1989-92.

16. Arshad AR. Parotid swellings: report of 110 consecutive cases. Med J Malaysia 1998;53:417-22.

17. Batsakis JG: Non-neoplastic diseases of the salivary glands, chap 3,Tumors of the Head and Neck, 2nd Ed. Baltimore, Williams & Wilkins,1979

18. Treatment of complications of parotid gland surgery. MARCHESE-RAGONA, C. DE FILIPPIS, G. MARIONI, A. STAFFIERI Department of Otolaryngology-Head Neck Surgery, University of Padua, Padua, Italy; ACTA OTORHINOLARYNGOL ITAL 25, 174-178, 2005

19. Staffieri A, Marchese Ragona R, de Filippi, s C, Tugnoli V. *Management of parotid fistulae and sialoceles with botulinum toxin.* Otolaryngol Head Neck Surg 1999;121:P240-1.

20. Washington manual of surgical pathology 1st Edition, Chapter 6, Salivary Glands; James S. Lewis Jr., Elise L. Krejci

21. Sabiston text book of surgery 18th edition

22. Sonographically Guided Core Biopsy of A Parotid Mass : David C. Howlett Leon J. Menezes, Khari Lewis, Andrew B. Moody, Nick Violaris, Michael D. Williams

23. Master of surgery, Joseph E Fisher, 5th Edition, Lippincott Williams & Wilkins; Volume I ; Head and Neck, The Parotid Gland

24. Salivary gland tumors - our experience Nitin m. Nagarkar, sandeep bansal, arjun dass, surinder k. Singhal, harsh mohan; I*ndian journal of otolaryngology and head and neck surgery vol. 56 no. 1, january - March 2004*

25. Retrospective Evaluation of Parotidectomy Cases Aydın ACAR1, Adil ERYILMAZ1, Melih ÇAYÖNÜ1, Halit AKMANSU1, Celil GÖÇER1, Bengi ARSLAN MUTLU1, Hayriye KARABULUT; Eur J Surg Sci 2010;1(2):47-52

26. Correlation between fine needle aspiration biopsy and histologic findings in parotid masses. Personal experience ; A.M. CONTUCCI et al, ACTA OTORHINOLARYNGOL ITAL 2003,23:314-318

PROFORMA

NAME AGE & SEX IP No.

OCCUPATION SOCIO-ECONOMIC STATUS

ADDRESS

HISTORY

PRESENTING HISTORY:

SWELLING DURATION DISCHARGE PAIN

SYMPTOMS OF FACIAL NERVE INVOLVEMENT

OTHER

PAST:

EXPOSURE TO RADIATION TREATMENT FOR SIMILAR LESION

PERSONAL

ALCOHOL SMOKING TOBACCO/ BETEL NUT CHEWING

EXAMINATION:

GENERAL EXAMINATION :

BUILD & NUTRITION

ANAEMIA CLUBBING LYMPH NODE ENLARGEMENT

LOCAL EXAMINATION:

NUMBER SITE SIZE SHAPE SKIN SURFACE

INDURATION

WARMTH TENDERNESS CONSISTENCY MOBILITY

BI-DIGITAL PALPATION

FACIAL NERVE INVOLVEMENT

REGIONAL LYMPH NODE ENLARGEMENT

INVESTIGATIONS:

FNAC REPORT

BLOOD INVESTIGATIONS : Hb% ESR TC & DC

RADIOLOGICAL:

USG SWELLING

CT HEAD AND NECK

TREATMENT

SURGERY

OTHER MODALITY USED

POST – OPERATIVE COMPLICATIONS IF ANY AND MANGEMENT

HISTO-PATHOLOGICAL REPORT:

Name	Age & Sex	IP No.	Prev Sx	Side	Size	Past History	Habits	Duration	VII Nerve Involvement	Mobility	Discharge	Skin	Nodes	Pain	Deep Lobe	FNAC	HPE	Surgery	Other Findings	Other Rx	P.O. Complications
Jayaeshwari	55/F	232	no	left	6x5cm	nil	nil	3 years	nil	mobile	nil	free	level IB,II, III, IV,V	nil	nil	ChSA	NHL	Lt. RP + RND	muscle infiltration	CHOP Regimen	left VC plasy
Palaniammal	38/F	289	no	left	4x5cm	nil	tobacco chewing	3 months	nil	mobile	nil	free	nil	nil	nil	MEC	MEC- LG	Lt TCP	nil	nil	seroma
Mahalakshmi	48/F	258	no	left	3x3cm	nil	nil	1 year	nil	mobile	nil	free	level II	nil	nil	RA	RA	Lt SP	nil	nil	nil
Karthiga	21/F	269	yes	left	4x3cm	prev swelling+	nil	6 months	nil	fixed	nil	scar +, skin scarred	level II	nil	involved	ACCA	AdCA	Lt ERP+Level II ND	facial nerve trunk encased	P.O. RT	VII N
Palaniammal	60/f	303	yes	right	4x5cm	abcess drainage-15yrs ago	tobacco chewing	3 months	nil	mobile	nil	free	nil	yes	involved	positive for malignancy	U.CA	Lt. ERP	infiltration+, zygomatico-temporal	P.O. RT	VII N
Meenambal	75/F	52	no	left	4X6cm	nil	tobacco chewing	6months	nil	fixed	nil	involved	Level II,III,IV,V	yes	nil	s/o malignancy	Ad.CCA	Lt. TCP Lt. RND	muscle infiltration	P.O. RT	VII N
Ramasamy	62/M	652	yes	right	5x7cm	prev swelling+	smoking	8months	nil	mobile	nil	involved	level II	nil	nil	CXPA	MPA	Rt TCP	infiltration to muscle, , all margins +ve	P.O. RT	seroma, flap necrosis
Jayalakshmi	50/F	602	yes	right	4x5cm	prev swelling+	nil	1 year	yes	mobile	nil	free	nil	nil	nil	ACCA	MEC- LG	Rt CP	margins free, node-reactive hyperplasia	P.O. RT	seroma, facial palsy
Munusamy	72/M	386	no	right	6x7cm	nil	nil	1 month	yes	nil	yes	ulceration	level IB,IA, III	nil	nil	acute supurative inflammation	MEC- LG	Nil		P.RT	nil
Vasantha	65/F	1418	no	left	7x7cm	nil	nil	1 1/2 years	yes	nil	nil	fixed	level IB,II,	yes	nil	MEC	AdCA	Lt. ERP+ Lt. RND	infiltration, arterial encasement	P.O. RT	Facial nerve palsy
Ramasamy	70/M	55	no	left	8x6cm	nil	tobacco chewing	1 year	yes	fixed	nil	free	nil	nil	nil	probably mucoepidermoid Ca	MEC- HG	nil	nil	P.RT	nil
Citi Babu	64/M	1231	yes	right	5x3cm	nil	nil	8months	nil	nil	nil	scar	nil	nil	nil	ACCA	cystic neoplasm of parotid	Rt. CP	nil	P.O. RT	nil
Guna Sundari	26/F	1327	yes	right	5x2cm	nil	nil	7yrs	nil	mobile	nil	NIL	level II	nil	nil	MEC	MEC-LG	Rt TCP	lower facial nerve trunk involved	P.O. RT	Facial nerve palsy
Ravi	45/M	1240	no	right	12x7cm	nil	smoking	6months	nil	fixed	nil	free	nil	yes	nil	CXPA	CXPA	nil		P.RT	Facial nerve palsy
Durga Devi	17/F	18419	no	right	5x4cm	nil	nil	1week	nil	mobile	nil	NIL	nil	yes	nil	abscess		Rt. I&D	nil	nil	nil
Kanagavalli	37/F	14375	no	left	6x4cm	nil	nil	10 days	nil	mobile	nil	nil	nil	yes	nil	abscess		Rt. I&D	nil	nil	nil

Jayalakshmi	39/F	12797	yes	right	6x5cm	nil	nil	1 1/2 years	nil	mobile	nil	nil	nil	nil	nil	PA	PA	Rt. SP	nil	nil	nil
Dhayalan	63/M	9176	no	right	6x7cm	nil	nil	10months	nil	mobile	nil	nil	nil	nil	nil	MEC	MEC-HG	Rt. TCP+RND	nil	P.O. RT	seroma, flap necrosis
Jayalakshmi	39/F	11147	no	right	5x6cm	nil	nil	1 year	yes	mobile	NIL	NIL	nil	nil	nil	ACCA	ACCA	Rt TCP	nil	P.O. RT	seroma, facial palsy
Arun Kumar	16/M	11952	no	left	3x4cm	nil	nil	6months	nil	mobile	nil	NIL	nil	nil	nil	lipoma	lipoma	Lt. Ebx	nil	nil	nil
Saroja	35/F	13343	no	left	2x2cm	nil	nil	1year	nil	mobile	nil	NIL	nil	yes	nil	PA	PA	Lt. SP	nil	nil	nil
Deepa	16/F	20123	no	left	3x4cm	nil	nil	10days	nil	nil	nil	nil	nil	yes	nil	abscess		Lt. I&D	nil	nil	nil
Mosaraj Rathinam	54/M	18678	no	right	5x6cm	nil	smoking	3 months	nil	mobile	nil	nil	nil	yes	nil	abscess	chronic abscess	Rt.SP	nil	nil	nil
Appandu	65/M	24950	yes	right	6x7cm	nil	nil	3 months	nil	mobile	nil	scar+	nil	nil	nil	PA	PA	Rt.SP	nil	nil	nil
Matheena Beevi	63/F	17580	no	right	6x4cm	nil	nil	7 months	nil	mobile	nil	NIL	nil	nil	nil	PA	PA	Rt.SP	nil	nil	Facial nerve palsy
Mohideen	50/ M	25629	no	left	8x6cm	nil	smoking	2 years	nil	mobile	nil	nil	nil	nil	nil	WT	WT	Rt.SP	nil	nil	seroma
Govindaraj	42/M	23032	no	right	5x4cm	nil	nil	9months	nil	mobile	nil	nil	nil	nil	nil	PA	PA	Rt.SP	nil	nil	nil
Rajagopal	57/M	1576	no	left	6x5cm	nil	nil	6months	nil	mobile	nil	NIL	nil	nil	nil	PA	PA	Lt.SP	nil	nil	nil
Chinnamal	55/F	1657	no	left	6x4cm	nil	nil	1 year	nil	mobile	nil	NIL	nil	nil	nil	PA	PA	Lt.SP	nil	nil	nil
Satya	35F	12697	no	right	5x4cm	nil	nil	8 months	nil	mobile	nil	nil	nil	nil	nil	PA	PA	Rt.SP	nil	nil	nil
Haridas	42/M	24561	no	right	4x3cm	nil	nil	7 months	nil	mobile	nil	NIL	nil	nil	nil	cystic aspirate	parotid cyst	Rt.SP	nil	nil	seroma, parotid fistula
Malliga	34/F	3568	no	left	3x4cm	nil	nil	6 months	nil	mobile	nil	nil	nil	nil	nil	PA	PA	Lt.SP	nil	nil	parotid fistula
thirunavukarasu	70/M	13321	no	right	6x4cm	nil	nil	8 months	yes	fixed	nil	fixed	Level II,III,IV	nil	nil	MEC	MEC-HG	nil		P.RT	nil

Elumalai	29/M	1987	no	right	3x4cm	nil	smoking	6 months	nil	mobile	nil	NIL	nil	nil	nil	PA	PA	Rt.SP	nil	nil	nil
Velammal	60/F	15667	no	left	5x6cm	nil	nil	1 year	nil	mobile	nil	NIL	nil	nil	nil	PA	PA	Lt.SP	nil	nil	nil
Devaki	32/F	11451	no	right	4x3cm	nil	nil	5months	nil	mobile	nil	NIL	nil	nil	nil	PA	PA	Rt.SP	nil	nil	seroma
Dhanapal	49/M	12891	no	right	5x3cm	nil	nil	7 months	nil	mobile	nil	NIL	nil	nil	nil	PA	PA	Rt.SP	nil	nil	nil
Amutha	28/F	23013	no	left	4x3cm	nil	nil	8 months	nil	mobile	nil	NIL	nil	nil	nil	PA	PA	Lt.SP	nil	nil	nil
Janaki Raman	33/M	30012	no	right	6x4cm	nil	nil	1 1/2 years	nil	mobile	nil	NIL	nil	nil	nil	PA	PA	Rt.SP	nil	nil	nil
Jayanthi	47/F	23971	yes	left	4x3cm	nil	nil	6 months	nil	mobile	nil	scar +	nil	nil	nil	PA	PA	Lt.SP	nil	nil	seroma
Chellamai	52/F	1784	no	left	5x4cm	nil	nil	9months	nil	mobile	nil	NIL	nil	nil	nil	PA	PA	Lt.SP	nil	nil	nil
Kumar	41/M	16671	no	right	3x4cm	nil	nil	6 months	nil	mobile	nil	NIL	nil	nil	nil	cystic aspirate	PA	Lt.SP	nil	nil	seroma
Kamala	65/F	3224	no	right	5x4cm	nil	tobacco chewing	1 year	nil	mobile	nil	NIL	nil	nil	nil	PA	PA	Rt.SP	nil	nil	nil
Kodeshwari	50/F	5030	no	right	4x3cm	nil	nil	1 1/2 years	nil	mobile	nil	NIL	nil	yes	nil	C.SA	C.SA	Rt.SP	nil	nil	nil
Bahadur	77/M	3903	no	right	6x5cm	nil	nil	1 year	nil	mobile	nil	NIL	nil	nil	nil	PA	PA	Rt.SP	nil	nil	nil

KEY TO MASTER CHART

CSA	-	Chronic Sialadenitis
MEC	-	Mucoepidermoid Carcinoma
PA	-	Pleomorphic Adenoma
RA	-	Reactive adenitis
ACCA	-	Acinic cell tumor Carcinoma
Ad. CA	-	Adenoid cystic Carcinoma
UCA	-	Undifferentiated carcinoma
CxPA	-	Carcinoma ex pleomorphic adenoma
WT	-	Warthin's tumour
Inf	-	Inflammatory
PRT	-	Palliative RT
PORT	-	Postoperative RT
RP	-	Radical parotidectomy
TCP	-	Total conservative parotidectomy
SP	-	Superficial parotidectomy
ERP	-	Extended radical parotidectomy
CP	-	Completion parotidectomy
I&D	-	Incision and drainage
RND	-	Radical Neck Dissection

INSTITUTIONAL ETHICAL COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Ref.No.1463/ME-1/Ethics/2012 Dt:08.05.2012.
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "A Study on parotid swellings" submitted by Dr.Anu Ramesh, MS(GS), PG Student, KMC, Ch-10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

GOVT. KILPAUK MEDICAL COLLEGE
26 JUL 2012
Kilpauk, Chennai-10.

CHAIRMAN, 26/7/12
Ethical Committee
Govt.Kilpauk Medical College,Chennai